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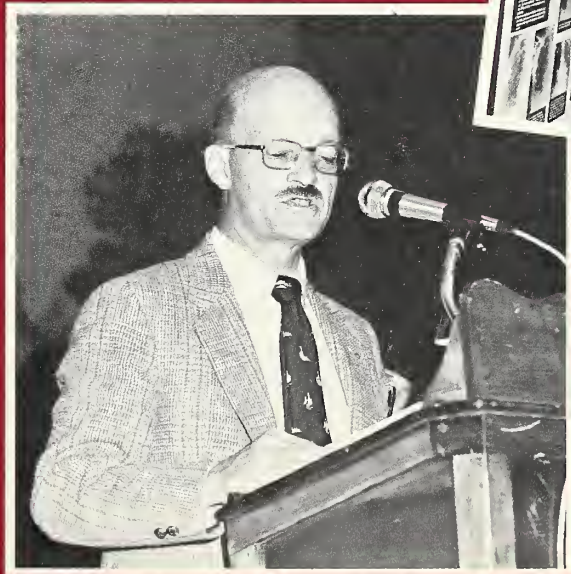
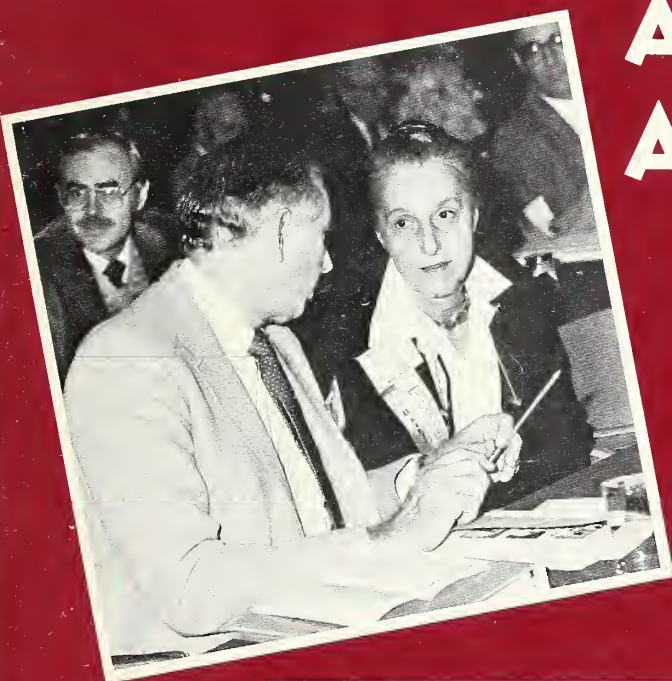
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The Award Winning Publication of The Medical Society of Virginia

## AT THE ANNUAL MEETING







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### KEY TO ABBREVIATIONS

b—book reviews	*—medical article
cr—committee report	m—memoir
ed—editorial	nb—news bureau
h—history	pv—point of view
le—letter to editor	s—speech
ml—medicine and law	sa—special article
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## Delegates ask change in Natural Death Act

By Bowen Hosford, JD

VIRGINIA'S NATURAL DEATH ACT, which went into effect only six months ago, was the object of action by The Medical Society of Virginia's House of Delegates at the annual meeting late last year, and when the 1984 General Assembly convenes this month, the Society's legislative liaison will be working to implement that action.

The delegates' action arose from a recommendation of the MSV Legislative Committee "to exempt from the Act's coverage the situation where an incompetent patient without a declaration suddenly becomes terminally ill and no family is available to make a decision about withholding life-prolonging procedures."

In recommending the change, the Legislative Committee stated that the Act could lead to inappropriate treatment of some patients. "The Natural Death Act can be read to require that in all instances of withholding life-prolonging procedures from a terminally ill patient, the witnessing and authorization provisions of the Act must be met," the committee commented. "There are situations in which this may be impossible, creating a situation where, in the absence of a declara-



In the House of Delegates at the annual meeting: from left, Dr. Haskins Ferrell, Jr.; Dr. Emory F. Hodges, Jr.; Dr. Robert H. Anderson; and Dr. William E. Sheely, all of the Alexandria Medical Society.

tion or authorization, extraordinary measures are used with every patient even when there is no hope of recovery."

The section of the Act to which committee members objected refers to terminally ill patients who are comatose, incompetent, or unable to communicate. The section presumes the patients haven't earlier signed declarations that forbid life-prolonging procedures or that name someone else to make decisions in such circumstances. Lacking such a declaration, the attending physician is to consult and agree with two family members, if they are reasonably available, in front of two witnesses, before the procedures are withheld. Family

members in order of preference are the patient's spouse, a majority of adult children, the patient's parents, or other nearest relative. If one of them isn't reasonably available or refuses to decide, the physician is to consult the next one down. Any guardian or committee that a judge had appointed would take precedence over family members.

Granted, it is doubtful that those requirements apply in every instance of withholding life-prolonging treatment from a terminally ill, incompetent patient, for there is an escape clause in another part of the Act that preserves "existing rights and responsibilities . . . which a health care provider . . . may have. . . ." Among their rights and



# NEWS BUREAU

responsibilities, physicians recognize that terminally ill and irreversibly comatose patients have different needs than others. The language of the section on incompetent patients, however, when read alone, is far-reaching.

For example, it apparently affects the writing of "no-code orders," also known as "DNR" (Do Not Resuscitate) or "No M-SET" (No Medical-Surgical Emergency Team). It has had a chilling effect, at least initially, on the writing of such orders for incompetent patients at Fairfax Hospital in Northern Virginia, where it has been discussed in the hospital's Ethics Forum chaired by Eugene W. Overton, MD.

A forum member, Ian Shenk, MD, said physicians will be uncertain whether they consulted the right people on the family list or had the right number of witnesses. "I'm afraid that where the legislators had a desire to allow people to die with dignity, fewer people are going to be allowed to die with dignity because of the insecurity of physicians in knowing whether they have met every requirement," Dr. Shenk said.

Before the Act was passed, Dr. Shenk chaired an Ethics Forum subcommittee that drafted "No M-SET" guidelines for Fairfax Hospital. They have been approved by the hospital's departments and are being considered by trustees. The guidelines are modeled on those at the Hennepin County, Minnesota, Medical Center, a teaching hospital associated with the University of Minnesota Medical School, and

contain the following key paragraph: "When the patient is competent, the No M-SET decision will be reached consensually by the patient and physician. When the patient is incompetent, this decision will be reached consensually by appropriate family members and the physician."

That paragraph takes into account today's medical, ethical and legal climate. It may be suggested that the Virginia Natural Death Act, in going further, overprotects incompetent patients. For example, legislators added the two-witnesses requirement after a General Assembly subcommittee drafted the Act. The Medical Society of Virginia's legal counsel, Allen Goolsby, believes they added the requirement not because they distrusted physicians but because they feared that family members might "do wrong."

Irrespective of the legislators' motives, it appears prudent for physicians under usual circumstances to agree with two family members in front of two witnesses before writing no-code orders.

"I believe that the Act does apply to no-code orders," Mr. Goolsby commented. "For this reason, I strongly recommend compliance with the requirements of the act if, under the circumstances, it is practical to do so."

The next question is what to do when it isn't practical to follow the procedures. For example, physicians worry that the Act might inhibit decisions in emergencies, in which it would be cruel to resuscitate

a patient but in which the family might not be available. Granted, the Act isn't meant to regulate good medical practice in every situation, and it contains that escape clause, so one is inclined to think that a court would be sympathetic to a physician's non-compliance in such circumstances. The chairman of the General Assembly subcommittee that drafted the Act, Bernard S. Cohen, of Alexandria, believes that fears are overstated. He comments, "It is ironic that some physicians have misinterpreted some parts of the Act and ignored other important parts, to the end that they now are reluctant to enter a 'no-code' order for a comatose patient arriving in the emergency room."

Only a court decision, however, would settle the question with certainty.

Whether Virginia legislators will modify the Act after only a few months' experience with it remains to be seen. Generally, they have been reluctant to amend new acts in less than a year.

*Journalist and lawyer Bowen Hosford is Freedom of Information officer for the National Institutes of Health and a member of the Ethics Forum at the Fairfax Hospital.*



# WITH THE MEDICAL SOCIETY OF VIRGINIA AT THE ANNUAL MEETING



▼ Dr. Lawrence K. Monahan, Roanoke (left), and Dr. James G. Brown, Alexandria (right), check the credentials of Dr. James A. Shield, Jr., of the Richmond Academy.



▼ Mrs. Gerald Weitzman, President, the Auxiliary. Her report of the Auxiliary's activities appears on page 35.







▼ Dr. James B. Kenley, State Commissioner of Health, reports to the delegates.



▲ Dr. Harold L. Williams delivers his Presidential Address, which you will find on page 32 of this issue. Dr. Williams appears at the microphone again in the picture at left of the House of Delegates in the Commonwealth Room at the Homestead.

▼ Dr. Harrison L. Rogers, Jr., Speaker of the AMA's House of Delegates (left), congratulates Dr. C. Barrie Cook after installing the Fairfax pathologist as The Medical Society of Virginia's new President.





▼ Medical Society of Virginia officers for 1983-1984: front row from left, Dr. Harry C. Kuykendall, Alexandria, President Elect; Dr. C. Barrie Cook, Fairfax, President; Dr. Anthony J. Munoz, Farmville, First Vice President; and Dr. Richard L. Fields, Fairfax, Speaker of the House; second row from left, Dr. Robert C. Green, Jr., Winchester, Second Vice President; Dr. Darrell K. Gilliam, Richmond, Third Vice President; and Dr. William H. Barney, Vice Speaker.







▲ Dr. Henry D. Patterson, Chesapeake, left, and Dr. T. K. Parthasarathy, Big Stone Gap.



▲ Dr. Joseph H. Callicott, Jr., left, and Dr. Jacques E. Botton, both of Lynchburg.

*Photographs of the annual meeting by Werner Gattinger*





▲ Dr. Austin R. Harrelson, Richmond. At rear, Dr. Henry H. Wilson, Jr., and Dr. Marcella F. Fierro, both also of Richmond.

▼ Dr. Stuart Ashman and Dr. John P. Clarke, both of Virginia Beach.



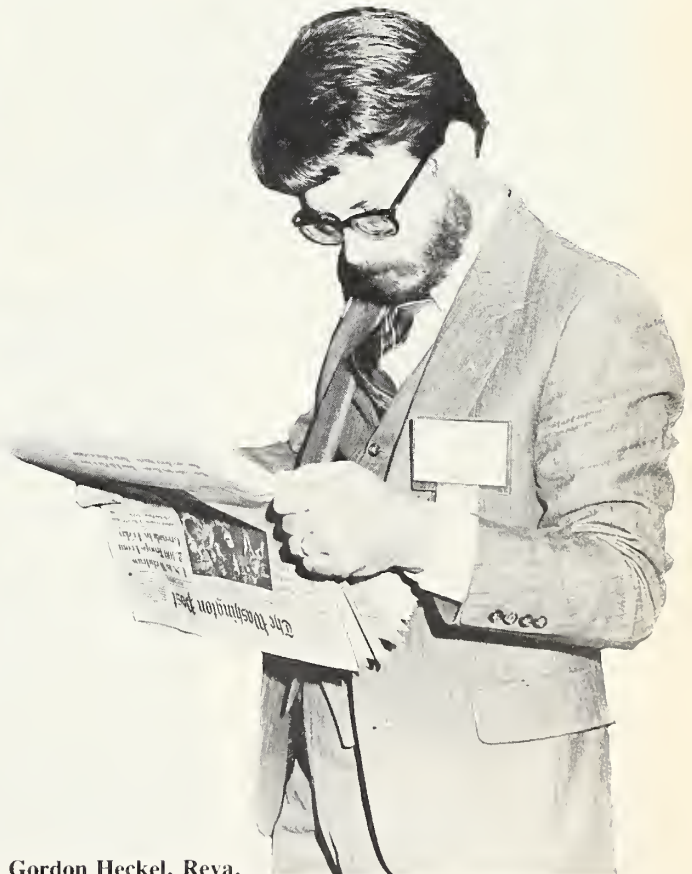
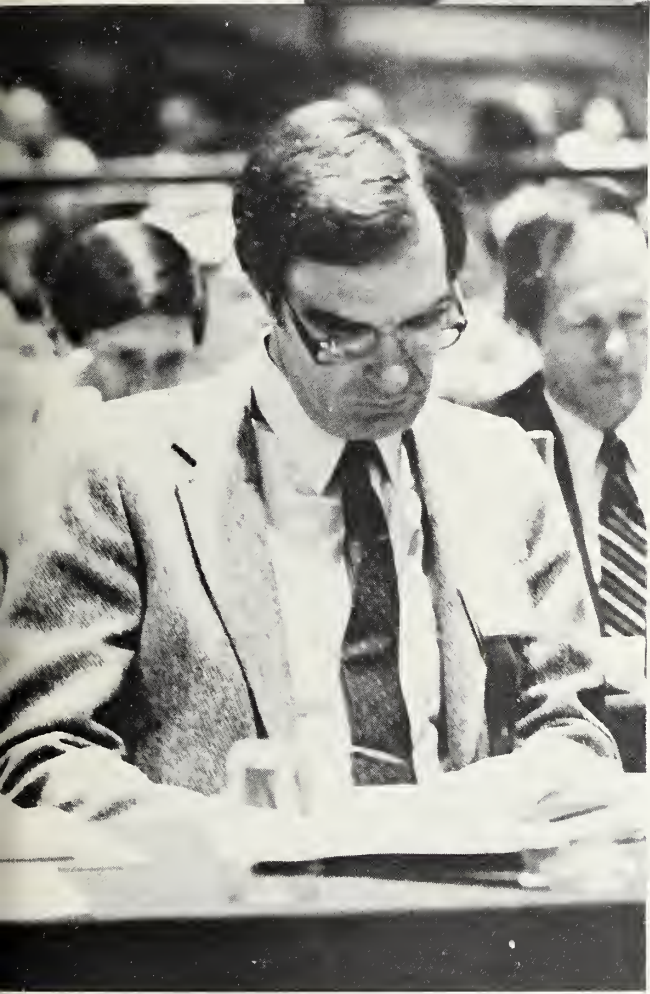
▼ Dr. Ronald F. Calkins, Dr. James L. Gaphery, and Dr. Richardson Grinnan, all of Richmond.







▲ Dr. John A. Owen, Jr., Dr. Ruth B. Weeks, both of Charlottesville; Dr. Eloise C. Haun, Woodstock, and Dr. William F. Tompkins, Charlottesville. At rear left, Dr. George E. Broman, Culpeper.



► Dr. C. Gordon Heckel, Reva.





▲ Dr. Albert J. Wasserman, Richmond, looks on as Dr. Phillip R. Thomason, Portsmouth, tries his hand at a computer case simulation in the exhibit sponsored by Dr. Wasserman on behalf of the Medical College of Virginia, where the programming is part of instruction in cost-effective care.

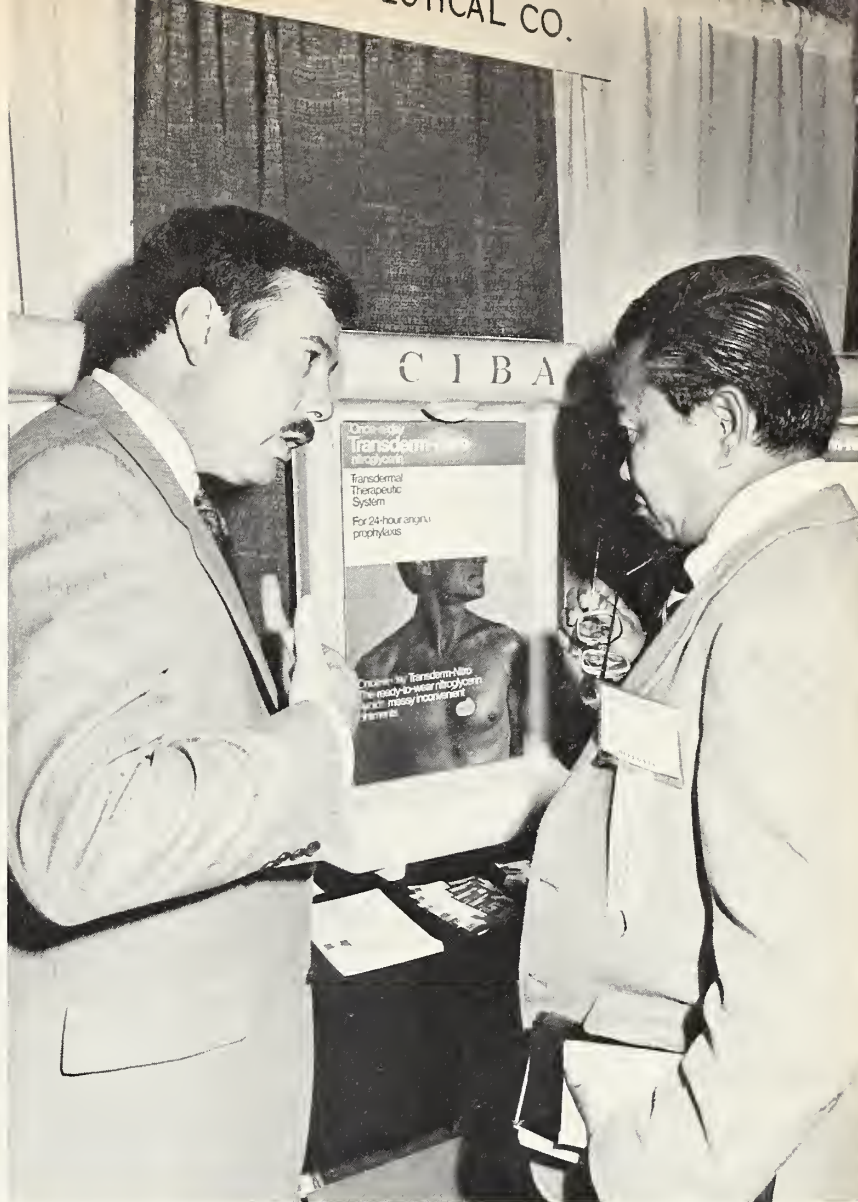


► Dr. Robert P. Trice, Richmond (right), stops by the Boots Pharmaceuticals booth. At left, rep Ted Lee.





▼ The first prize winner in the scientific exhibit section is the setting for, from left, Dr. Michael J. Moore, Roanoke; Dr. Clarence W. Taylor, Shaws-ville; and Dr. Wilson C. Merchant III, Richmond. Dr. Merchant and Dr. Wil- liam S. Tunner put together the blue ribbon exhibit, titled "Pediatric Blunt Renal Trauma."



▲ Dr. Francisco R. Barrera of Wood-bridge (right), ponders the message of rep Carey Mickel at the Ciba Pharma- ceuticals exhibit.



◀ Dr. George R. Smith, Jr., Shaws- ville (right), visits the Kremers-Urban exhibit and rep Sam Armstrong.

# THE HOUSE OF DELEGATES

At its session on November 5, 1983, at the Homestead in Hot Springs, the House of Delegates of The Medical Society of Virginia took the following actions. Dr. Richard L. Fields, Speaker, and Dr. William H. Barney, Vice Speaker, presided.

## HONORS

In the photograph on page 6 of this issue you will see the officers elected by the House of Delegates for 1983-1984.

The delegates also elected two new councilors, Dr. William W. Regan and Dr. John A. Owen, Jr., and one new vice councilor, Dr. Ashley Futral, Jr.

Dr. William J. Hagood, Jr., was named an AMA delegate, while Dr. Harold L. Williams joined the contingent of alternates, and Speaker Fields was designated to accompany the delegation to AMA meetings.

For a complete roster of the MSV leadership for 1983-1984, see page 14 of this issue.

To Dr. Williams, the meeting's presiding officer, the delegates awarded honorary active MSV membership; his Presidential Address, delivered at the House's first session, appears on page 32 of this issue.

To Governor Robb the delegates sent these nominations for 5th district representative to the State Board of Medicine: Dr. Edwin J. Harvie, Jr.; Dr. Gerald C. Burnett; and Dr. James E. Layton.

A Certificate of Distinguished Service was presented by Dr. Williams to Frank B. Hancock, Jr., veteran reporter for the *Roanoke Times & World-News*. Hancock was praised for his "professional excellence, accuracy in reporting medical affairs, and his role as a friend of the Roanoke Academy of Medicine and The Medical Society of Virginia."

From Speaker Fields, Dr. Duncan S. Owen, Jr., Associate Editor of *VIRGINIA MEDICAL*, received a framed certificate and a check for \$250 signifying the journal's recognition in the 1983 Sandoz Medical Journalism Award contest.

## LEGISLATIVE ACTION

### To Tax or Not to Tax?

The tax-exempt status of prepaid health plans was the subject of lively debate in the reference

committee hearing that preceded the session of the House. At issue: a 2.75% premium tax that insurance firms in Virginia are required to pay. A recommendation from the Legislative Committee called for the General Assembly to remove the exemption, and the reference committee finally voted to adopt it.

But in the House, after Past President W. Leonard Weyl rose to abjure the physicians to steer clear of the controversy, the delegates rejected the recommended position. Instead, they approved a substitute motion calling for the General Assembly to study "the effect of legislating tax-exempt status for insurance carriers, prepaid health care plans, and other private third-party payers, focusing on competition and health care costs."

### Consent and Cancer

Amid heated tempers and headlines, a proposal to mandate informed consent for breast cancer patients lost in last year's General Assembly, but proponents are said to be tuning up for a rerun in 1984. The delegates reiterated the Society's opposition of last year to any legislation that would require doctors to tell newly diagnosed breast cancer patients of treatment alternatives.

Separately, the House asked the Society to join the American Cancer Society in sponsoring education programs for physicians on options for breast cancer therapy.

### Transplants at Retail?

Sales of human organs for transplant purposes should be prohibited by state law in Virginia. That was the unequivocal message of a resolution that passed the House swiftly and unanimously.

### Liability Easements

Medical malpractice entered the delegates' deliberations through three resolutions. All were adopted. They asked Virginia's legislators to study

1. modification of the collateral source rule so that payments to a plaintiff by an independent source could be introduced as evidence in liability litigation;

2. institution of greater equality in the awards system, which now confers sums four times larger for injuries allegedly due to medical malpractice



than for similar injuries sustained in auto accidents;

3. placement of a "reasonable" cap on awards for pain and suffering in medical malpractice cases because the resulting higher insurance premiums contribute to rising costs.

### **Auto Safety**

Seat restraints for infants in motor vehicles was the object of legislation supported by The Medical Society of Virginia and passed by the 1982 General Assembly. This year, the MSV House decreed, the Society is to support a law that would require everyone, young and old alike, to be secured by safety devices when riding in autos of post-1968 manufacture.

The House also approved Society support of some minor changes in the law mandating child restraints to facilitate enforcement by the police.

Medical Society of Virginia support for the efforts of the Governor's Task Force to Combat Drunk Driving was endorsed by the delegates.

Adopted by the House was a resolution committing the Society's support for legislation "that would strengthen statutory penalties for driving a moving vehicle while under the influence of alcohol."

### **MD Reimbursement**

The House passed a resolution asking for legislation "which forbids prepaid medical and surgical plans from prohibiting a subscriber from assigning right to payment for covered services to the physician of the subscriber's choice."

### **Certificates**

In separate recommendations, the Legislative Committee asked for statutory certification of athletic trainers and respiratory therapists and voluntary certification of occupational therapists. The House approved.

### **Treating Patients**

The House adopted a resolution in support of legislation requiring immunization against rubella for all students born after 1963 who matriculate for "post-scholastic" studies at two- and four-year colleges and trade professional schools in Virginia.

Support for repeal of the statute that authorizes the administration of epinephrine in emergency situations was approved by the delegates. This law was passed on the assumption of a training program to teach lay persons how to administer epinephrine safely, but the American College of Emergency Physicians questioned such a program.

### **Award to Carmines**

To Dr. F. Ashton Carmines, Jr., Newport News orthopedist, went the 1983 Physician Award for Community Service presented each year at the banquet that closes The Medical Society of Virginia's annual meeting. A man of deep religiosity, Dr. Carmines has traveled the world on missions combining his faith and his profession and is identified with Medical Society of Virginia leadership. More about this in the February issue, plus a picture of 50-Year Club members at the meeting.

The delegates also voted to support an amendment to the Good Samaritan law to make clear that those administering epinephrine to a person diagnosed as being at risk are immune from liability.

### **Death and Dying**

A change in Virginia's Natural Death Act, passed with Society support by last year's General Assembly, was requested in action by the delegates. They asked for exemption for the situation in which "an incompetent patient without a declaration suddenly becomes terminally ill and no family is available to make a decision about the withholding of life-prolonging procedures."

### **Board Opposition**

Two legislative moves affecting Virginia's health regulatory boards are to come before the 1984 General Assembly, and both met opposition from The Medical Society of Virginia's delegates. They voted to oppose 1) the addition of citizen members to some of the boards without adding them to all; and 2) they opposed the transfer of the boards to the State Department of Commerce. For an editorial on this second proposal, see page 33 of this issue.

### **Rules and Regs**

The House went on record opposing legislation to regulate hospital rates in Virginia. Said the Legislative Committee of this recommendation, "Adding such a variable to the other pro-competition measures recently introduced would only add confusion" and would probably fail of the desired result.

The delegates registered approval of MSV support for legislation exempting home health care services from the certificate of need process.

The delegates also opposed changes in Virginia's commitment statute "which would subject the private sector to unnecessary regulation," but they



# Medical Society of Virginia Officers and Councilors

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President	C. Barrie Cook, MD
Past President	Harold L. Williams, MD
President Elect	Harry C. Kuykendall, MD
Vice Presidents	Anthony J. Munoz, MD; Robert C. Green, Jr., MD; Darrell K. Gilliam, MD
Speaker of the House	Richard L. Fields, MD      Vice Speaker, William H. Barney, MD
Councilors	1st District, William S. Burton, MD; 2nd, Russell D. Evett, MD; 3rd, William W. Regan, MD; 4th, H. Alan Bigley, MD; 5th, Glenn B. Updike, Jr., MD; 6th, William W. S. Butler, MD; 7th, John A. Owen, Jr., MD; 8th, Nicholas G. Colletti MD; 9th, J. Thomas Hulvey, MD; 10th, Leon I. Block, MD
Vice Councilors	1st District, William H. Sipe, MD; 2nd, John L. Dobson, MD; 3rd, C. M. Kinloch Nelson, MD; 4th, John W. Hollowell, MD; 5th, Gerald C. Burnett, MD; 6th, Robert L. Keeley, MD; 7th, A. Ashley Futral, Jr., MD; 8th, Ira J. Green, MD; 9th, James L. Patterson, Jr., MD; 10th, Donald S. Thorn, MD
Councilors Ex Officio	James B. Kenley, MD, State Commissioner of Health Edwin L. Kendig, Jr., MD, Editor, VIRGINIA MEDICAL
AMA Delegates	Michael A. Puzak, MD; William J. Hagood, Jr., MD; W. Leonard Weyl, MD; F. Ashton Carmines, MD; John A. Martin, MD; Raymond S. Brown, MD
Alternate Delegates	George M. Nipe, MD; Percy Wootton, MD; Charles M. Caravati, Jr., MD; Arthur A. Kirk, MD; H. C. Alexander III, MD; Harold L. Williams, MD
Executive Vice President	James L. Moore, Jr.      Emeritus, Robert I. Howard

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voted to support changes which would balance appropriately "the legal, medical and psychological needs of persons subject to the involuntary commitment process."

Adopted unanimously by the House was a recommendation calling for support of legislation to insure that "disclosure of confidential information to third-party payers relating to psychiatric treatment be minimized to the extent possible."

## GENERAL

### Public Service

The American Medical Association's Patient Medication Instruction Forms, or PMIs, generated a resolution pledging The Medical Society of Virginia to take "an active and aggressive position in support of the use of PMIs by Virginia physicians." The delegates unanimously approved.

Another resolution passed by the House without demurral asked that all public housing units be required to be equipped with smoke detectors and

all homeowners be strongly encouraged to install smoke alarms.

Care for the needy prompted a resolution, endorsed by the House, that committed The Medical Society of Virginia to "make the needy aware, through its central office and component societies, of the availability of medical care."

A resolution relating to Virginia's elderly asked the Committee on Aging to see if "the unique health-care needs of the elderly are met, now and in future, as the percentage of elderly in the population increases." The House approved.

### Practice Protocol

Electromyography was found by the House to constitute the practice of medicine, and it asked the Board of Medicine to limit its use to licensed, qualified physicians.

The House also asked the Board of Medicine to "license nurse midwives to practice only under the direct supervision of a physician qualified in obstet-



# VaMed INTERCOM

Published by The Medical Society of Virginia

## REPORT FROM THE AMA MEETING

The American Medical Association's House of Delegates met at its interim session in Los Angeles December 4-7, 1983. Preceding the session the AMA Section on Medical Staffs attracted close to 380 delegates to discuss the many problems pertaining to hospital staffs and to make recommendations to the House. The Student and Resident Sections also met and sent multiple resolutions to be acted on by the AMA delegates.

After a full day of reference committee hearings and two days of floor debate, the delegates

- sought more study of UCR AND INDEMNITY payments;
  - adopted policy on Joint Commission MEDICAL STAFF standards;
  - opposed SURROGATE MOTHERHOOD;
  - called for changes in the INSANITY DEFENSE;
  - asked more study of FOREIGN MEDICAL GRADUATES policy;
  - set guidelines for BREAST CANCER SCREENING;
- urged revision of TEFRA provisions.

The delegates recognized "the validity of a pluralistic approach" in the determination of third-party reimbursement for physicians' services and stated that continuing physicians' freedom to establish their own fees carries with it the responsibility to avoid financial hardships for the patient. The House called for continuing analysis of UCR AND INDEMNITY payment methods, to be reported at the June 1984 meeting.

Regarding the emotional issue of MEDICAL STAFF privileges for licensed limited practitioners,

three main facts were made clear during debate. First, many laws allow access to the hospital for licensed limited practitioners. Second, federal antitrust laws guarantee enforcement of state laws. Third, the JCAH must address privileges for licensed limited practitioners as guaranteed by these laws.

After long debate, first in the Hospital Medical Staff Section and then in reference committee and the House, the delegates agreed that a national policy would not be appropriate and adopted the "Alabama Resolution" which states: "Hospital admitting privileges should be granted in accordance with state law and with the criteria for standards of medical care established by the individual hospital medical staff." The delegates instructed the JCAH to heed this resolution.

On SURROGATE MOTHERHOOD, the delegates adopted a report of the Judicial Council, "Supreme Court" of the AMA, which considered the grave ethical, legal and psychological risks of paying a woman to bear a child for another. The report stated that "these arrangements do not provide a satisfactory reproductive alternative."

By an overwhelming vote, the AMA delegates called for a narrowing of the use of the INSANITY DEFENSE in criminal trials. The report of the Board of Trustees' Committee on Medicolegal Affairs calls for replacement of the present insanity defense with statutes that would permit a defendant to be acquitted on insanity grounds only if mental disease prevented him from commit-

ting the criminal act with the requisite state of mind, or "mens rea." Still calling for retention of the original insanity plea are the American Bar Association and the American Psychiatric Association.

Many federal statutes pertaining to FOREIGN MEDICAL STUDENTS were addressed in a report of the Council of Medical Education. The House voted to send this report back to the Council for further study and reconsideration. The questions of examinations and qualifications of FMGs are also to be studied.

Regarding BREAST CANCER detection, the House approved a report calling for early detection, increased education and screening programs, as well as treatment protocols, to increase the cure rate of women with breast cancer. Specifically, the report calls for more awareness of the importance of self-examination, patient education by physicians regarding self-examination, recognition of mammography as an effective screening device to detect early cancer, and cytological examination of fluid aspirated from breast cysts when clinically indicated.

Delegates urged the repeal of a section of the Tax Equity and Fiscal Responsibility Act, TEFRA, that could deny surgical assistant fees at teaching hospitals. The House also called for extending the utilization and quality of care provision of TEFRA to all non-military federal medical institutions.

In other legislative matters, the House reaffirmed the need for

public funding for abortions and urged "Good Samaritan" legislative relief for in-flight medical emergency care.

Many other events took place and many other actions were taken. Notably, the House heard an address by Secretary of Health and Human Services Margaret Heckler, who stated that the federal government is depending on physicians to protect their patients from abuses that could occur under Medicare's new DRG plan and other cost-control measures. If physicians don't accept that responsibility, they may be forced into a "financing system so centralized that all payers join together in 'negotiating' with physicians, leaving you the options of objecting to the payments rules, going on strike, or leaving the country."

Finally, AMA President Frank Jirka told the House that physicians must fight the federal government's attempt to require them to accept mandatory assignment from Medicare patients. Legislation for mandatory assignment is "an unprecedented move to limit medical care for the aged." Government is trying to "shift the blame to physicians who refuse to sign on to the mandatory payment bandwagon." Dr. Jirka called on physicians to be unified in meeting the challenge of making the American medical system more cost-effective. If physicians in different specialties battle with one another, he queried, who will stand with either side?

W. LEONARD WEYL, MD,  
AMA Delegate



rics with privileges in a JCAH-approved hospital in the same community in which the midwife practices." The resolution also advocated that all such deliveries take place in a licensed hospital so that the infant receives optimum care.

### Money

The delegates approved the submitted budget with only two amendments: one authorizing reimbursement of traveling expenses for intervenors in the Physicians' Health and Effectiveness Program, the other providing reimbursement for the President Elect for his expenses up to \$3,000. The entire budget is printed with this report.

### Third Parties

A resolution that referred to Report D of the AMA Council on Medical Service asked the AMA to "seriously consider recommending that third parties change to an indemnity system of payment for physician services, i.e., paying a set amount for services rather than some proportion of the UCR charge." The House adopted the resolution.

The delegates also adopted a resolution asking Medicaid officials for concurrent review of claims for reimbursement rather than the retrospective system now operative.

Another action passed by the House asked The Medical Society of Virginia to publicize "the detrimental effect on patients of insurance policies that provide inpatient coverage only if rendered in specified hospitals" and require the insured to transfer from his physician to a hospital staff doctor.

The Society was also asked, in a resolution passed by the delegates, "to bring to the attention of its members the advantages of looking directly to the patient or his family for payment of the physician's services rather than relying on third-party reimbursement."

### Mall Medicine

Affirming the reference committee's recommendation, the delegates voted No to a resolution that sought "to establish minimal voluntary standards for freestanding emergency care centers." The standards were to be worked out, the resolution proposed, by The Medical Society of Virginia in cooperation with other professional organizations.

A. G.

## Budget 1983-1984

Salaries	\$332,000
Printing, mailing services—	
membership	2,500
Stationery, supplies	12,000
Office equipment—repairs,	
replacements	16,000
Building maintenance, repairs	38,000
Telephone	12,000
Postage	8,500
Convention expense	20,000
Council, committee expenses	14,000
Travel expense	
AMA delegates	25,000
Executive Vice President, staff	8,000
President Elect expense	
allowance	3,000
President expense account	12,500
President expense allowance	15,000
Virginia Medical	110,000
Editor	2,400
Legal expense	97,500
Legislative expense	11,000
Walter Reed Commission	1,500
Auxiliary	3,500
Dues (affiliated organizations)	1,200
VaMPAC	10,000
Newsletter	6,000
Continuing medical education	8,000
Physicians' Health/	
Effectiveness Program	12,000
Miscellaneous	5,000
Computer operation, maintenance	7,000
Auditor	5,000
Employee benefits	
Retirement fund	70,000
Medical, life, disability insurance	19,000
Payroll taxes	21,000
Consultants	
Legislative, PR	79,000
Insurance—actuarial services	10,000
Special appropriations	
Virginia Council Health/	
Medical Care	12,000
Building fund	10,000
AMA-ERF	1,000
Scholarships—MCV, UVa, EVMS	6,000
Rural health	1,500
Student medical societies—	
MCV, UVa, EVMS	8,500
Other special appropriations	21,000
TOTAL EXPENSES	\$1,057,600

# Resistance to Ampicillin in *Hemophilus Influenzae* Meningitis

Carl W. Armstrong, MD, and Joanne E. Butler, RN, *Richmond, Virginia*

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Reports to the Virginia Department of Health of cases of meningitis caused by *Hemophilus influenzae* were reviewed for 1977-1981. Ampicillin resistance of the infecting organism was not a risk factor in any of the 5.1% fatalities, demonstrating the wisdom of chloramphenicol and ampicillin therapy for a suspected case of the disease until results of anti-microbial susceptibility tests are known.

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**A**MPICILLIN-RESISTANT isolates of *Hemophilus influenzae* type b were first reported in 1974<sup>1</sup>. The recognition that a certain percentage of invasive *H influenzae* isolates were resistant led to recommendations that chloramphenicol be added to the initial treatment regimen of all patients with a suspected severe infection due to *H influenzae*<sup>2</sup>. A survey of 45 large pediatric medical centers throughout the US during 1976-1977 found that the prevalence of ampicillin-resistant isolates among patients with meningitis or bacteremia was 4.5%. There was considerable variation, however, from one region of the country to another<sup>1</sup>. Susceptibility testing of *H influenzae* isolates, including those from respiratory sites, by

investigators in Huntsville, Alabama, and Boston, Massachusetts, revealed a progressive increase in the percentage of isolates found resistant to ampicillin during the years 1975 through 1977<sup>3</sup>.

In order to determine the impact of ampicillin resistance in this state, all cases of meningitis caused by *H influenzae* and reported to the Virginia State Health Department's Division of Epidemiology were reviewed. Virginia law requires the reporting of bacterial meningitis of all types.

## METHODS

Since 1977, the Division of Epidemiology has participated in a national bacterial meningitis and meningococemia surveillance system. Bacterial meningitis, therefore, has been a reportable disease and case reports have been received from physicians and hospitals throughout the state. A standard questionnaire was completed for each reported case and the following information was obtained: the

From the Division of Health Hazards Control, where Dr. Armstrong is Director, and the Division of Epidemiology (Miss Butler), Virginia State Department of Health, 109 Governor Street, Richmond VA 23219. Address correspondence to Dr. Armstrong.

Submitted 5-4-83.



patient's county of residence, age, sex, race, results of blood and cerebrospinal fluid (CSF) cultures, date of onset of illness, outcome, the serotype of the organism and its susceptibility to ampicillin. Copies of hospital records were requested for all reported cases. The Division of Vital Records and Health Statistics also supplied the Division of Epidemiology with copies of all death certificates on which *H influenzae* meningitis was listed as the cause, or a contributing cause, of death.

Tests for statistical significance were performed using either the chi-square test (chi-sq) or Fisher's exact test, one-tailed (FET).

### Definitions

A case of *H influenzae* meningitis was included in this analysis if the attending physician's clinical diagnosis was "meningitis", a lumbar puncture yielded CSF which demonstrated a pleocytosis and hypoglycorrhachia, and *H influenzae* was isolated from either CSF or blood (or both).

Most hospitals reported ampicillin susceptibility based on the results of a Kirby-Bauer disk test. Isolates reported to be intermediately sensitive were not counted. If the results of both disk susceptibility testing and testing for beta-lactamase production were reported, and if there was a discrepancy between these two tests (few were noted), then the beta-lactamase test was used to determine susceptibility.

## RESULTS

From 1977 through 1981, 406 cases of *H influenzae* meningitis were reported. There were 217 males and 189 females. Based on 1980 census data, the crude incidence rate per year for the five-year period was 1.52 per 100,000 population. For whites, this rate was 1.40, and for non-whites it was 1.67. The organism was isolated from the CSF of 404 cases. In one case the result of the CSF culture was unknown, and in another case, in which the patient had received prior antibiotics, it was negative. The isolate serotype was known for 194 cases, and of these, 190 (98%) were type b. Of the remaining four, one was type a, one type c, one type d, and the last could not be typed.

Isolates from 88 cases (22%) were found to be resistant to ampicillin by disk susceptibility testing, broth dilution minimum inhibitory concentration (MIC), or by testing for beta-lactamase production. None of the isolates was reported to be resistant to chloramphenicol.

Results of testing for beta-lactamase production

were reported for only 14% (56/404) of the sensitive and resistant isolates. The trend, however, was for increasing use of this test during recent years; the percentage of all isolates so tested rose from 2% in 1977 to 24% in 1981.

### Comparison of Cases by Ampicillin-Sensitive and -Resistant Organisms

Testing for the production of beta-lactamase was more likely to have been performed on resistant isolates (18/88) than isolates (38/318) found to be sensitive ( $p < .05$  by chi-sq).

There was no apparent increase or decrease in the proportion of isolates resistant to ampicillin during the period of study. The percentage of isolates resistant were as follows, per year: 1977, 20%; 1978, 22%; 1979, 16%; 1980, 25%; and 1981, 23%. The percentage of isolates found resistant in 1977 was not statistically different from that in 1981.

The percentage of isolates resistant to ampicillin was determined for each of the five Health Systems Agency regions: region I (northwest), 26%; region II (northern), 22%; region III (southwest), 25%; region IV (central), 19%, and region V (eastern), 17%. Neither the highest (26%), nor the lowest (17%) regional prevalence of resistance was significantly different from the remainder of the state.

The age distribution of cases with resistant isolates was almost identical to that of cases with sensitive isolates; 56% of cases with resistant isolates were under one year of age, and 95% were 5 years of age or younger. For cases with sensitive isolates, the corresponding percentages were 51% and 94%, respectively.

The seasonal distribution was likewise similar for both groups; 65% of cases due to resistant organisms had onsets of illness during the six-month period October through March, while for those due to sensitive organisms, the percentage was 60%.

A majority of cases caused by resistant organisms had associated bacteremia (79%). Bacteremia was not significantly less common (74%) among those infected by sensitive organisms. Likewise, a fatal outcome was no more likely for cases caused by resistant isolates (4.6%) than for cases caused by sensitive isolates (5.3%).

### Meningitis in Adults

Fifteen adult cases were reported. Although adults were arbitrarily defined as those persons 16 years of age or older, the median age of the reported cases was 56 years. Most of the adult cases were reported towards the end of the study period; there were none reported in 1977, one in 1978, two in 1979, four in 1980, and eight in 1981. The percent-

age of cases in adults was significantly higher ( $p < .05$  by chi-sq) for 1980-1981 (5.5%) than for 1977-1979 (1.6%).

There was no significant difference in the prevalence of resistant isolates for adults (20%) and children (22%).

The case-fatality rate for adults was 20% (3/15), significantly higher than the rate of 4.5% (17/377) for children ( $p = .035$  by FET). The overall case fatality rate was 5.1%.

Five of the adult cases had histories of conditions which might have predisposed them to an invasive *H influenzae* infection. One had had a neurosurgical procedure (frontal lobotomy), one had alcoholic liver disease, one had hypogammaglobulinemia of unknown etiology, one was diabetic, and one had had pneumococcal meningitis several years previously.

Seven adults developed symptoms of meningitis without any preceding focal symptoms, whereas the illness for the remaining eight began with otitis media in four, an upper respiratory infection in three, and sinusitis in one. None had pneumonia.

## DISCUSSION

The calculated incidence of *H influenzae* meningitis found in this study, 1.52 cases per 100,000 population, is undoubtedly lower than the true incidence, because complete reporting of all cases in a passive surveillance system is almost never achieved. This incidence rate for Virginia does, however, closely match the rate (1.24/100,000) derived from surveillance in 38 states participating in the national bacterial meningitis study<sup>4</sup>. Incidence rates which were derived from studies using more intensive case-finding techniques in localized areas of the country have generally been two to three times higher than the rate observed here<sup>5-8</sup>. The number of cases reported per year increased steadily throughout the period of this study. A major factor causing this increase was undoubtedly an improvement in the completeness of reporting with time. Because of this, it was not possible to determine if the true incidence of *H influenzae* meningitis has been increasing. Investigators in some areas of the country have suggested that there has been a true increase in the incidence of *H influenzae* meningitis, while others have maintained that the reported increases were artifactual<sup>9</sup>.

Of reported *H influenzae* meningitis in Virginia, 22% was caused by organisms found to be resistant to ampicillin. Although this prevalence is similar to the 18% overall prevalence found for 38 states<sup>4</sup>,

both rates may be artificially inflated by preferential reporting of cases caused by ampicillin-resistant strains. Although differences in the prevalence of resistance have been observed for various parts of the US,<sup>1</sup> we found no significant differences between geographic regions within Virginia.

Susceptibility tests employing the detection of beta-lactamase production were more likely to have been performed on ampicillin-resistant isolates than on sensitive isolates. This finding was not surprising, since many laboratories used the beta-lactamase test for confirmation of ampicillin resistance detected by other methods. In addition, laboratories which used the beta-lactamase test routinely, not just for confirmation, may have detected more resistant isolates than laboratories using standard disk diffusion procedures. *H influenzae* is known for its unpredictable "behavior" during susceptibility testing by disk diffusion, making this test less reliable than the beta-lactamase test. This unreliability stems from two factors. First, the results of disk diffusion testing of *H influenzae* are heavily dependent on the size of the inoculum<sup>10</sup>. Second, the media normally employed for disk diffusion testing, Mueller-Hinton agar, is nutritionally inadequate for supporting the growth of *Hemophilus*, while the use of chocolate agar, which is nutritionally adequate, yields unreliable results<sup>11</sup>.

Although a higher percentage of adult cases was noted during the most recent years of study, this may be an artifact of preferential reporting of adult cases. *H influenzae* infections in adults have received considerable attention recently in the medical literature<sup>12,13</sup>. Much of the renewed interest has centered around trying to explain why certain adults are at risk for this infection. For many years, it was widely believed that the vast majority of adults possessed protective antibody against *H influenzae* type b<sup>14</sup>. Adult cases of invasive disease, including meningitis, have been attributed in many cases to underlying diseases which might have compromised host defenses<sup>15</sup>. If this were the case, one might expect a fatal outcome to be more common for cases in adults than for cases in previously healthy children. Indeed, studies comparing case fatality rates for adults with those for children have found, as in our study, that adult rates were much higher<sup>6,16</sup>. Recent evidence suggests, however, that not all adults possess antibody against *H influenzae*, type b<sup>17</sup>, and furthermore that the presence of antibody, even though bactericidal in vitro, may not correlate with protection in vivo<sup>12</sup>. This may explain why two-thirds of the reported adult cases in our study had no obvious predisposing



condition. Some of the previously healthy adult cases may also have been at increased risk for infection because of their genetic constitution<sup>13</sup>, but whether or not this would also increase their chances of dying from their infections is not known. Although one study found that the prevalence of ampicillin-resistance was higher among pediatric than among adult cases<sup>18</sup>, no such difference was observed in our study.

No attempt was made in this study to identify secondary cases, if any, arising out of contact with the reported cases. None of the reported cases, however, was stated to have been a known contact of another case.

Because death certificates were used to improve the finding of fatal cases, while cases that survived were found by passive surveillance alone, the case fatality rates were undoubtedly inflated artificially. In spite of this, the childhood case fatality rate (4.5%) was relatively low for this serious infection, testifying to the efficacy of modern medicine, and compares with the 80% mortality observed in the pre-antibiotic era<sup>16</sup>. Additionally, the fact that ampicillin resistance of the infecting organism was not a risk factor for a fatal outcome in this study demonstrates the wisdom of the widespread practice of using chloramphenicol and ampicillin for the treatment of suspected *H influenzae* meningitis until the results of anti-microbial susceptibility tests are known.

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# Clinicopathology Conference: Diarrhea and Abdominal Pain

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From the Departments of Medicine and Pathology,  
University of Virginia School of Medicine  
Case Presentation by Michael E. Williams, MD  
Clinical Discussion by Edward W. Hook, MD  
Pathology Discussion by Joseph D. Schwartzman, MD

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## PRESENTATION OF CASE

DR. WILLIAMS: A 53-year-old disabled truck driver was admitted to the University of Virginia Hospital for evaluation of diarrhea and abdominal pain. He had been in his usual fair state of health until two weeks before admission, when he experienced the onset of sharp pain in the right lower quadrant and frequent diarrhea. The abdominal pain was persistent and nonradiating and increased during eating and defecation; the patient experienced some pain relief after each bowel movement. The diarrhea was described as voluminous, foul-smelling, foamy, watery, and yellow to milky white in color; the stools were not bloody and were noted to float. Over a period of a week, his stools increased in frequency to every two or three hours and became associated with tenesmus. His abdominal pain gradually worsened until it interfered with his sleep. He became anorectic and nauseated but did not vomit.

Edited by Dr. Williams; Erik Hewlett MD; and Marcia Day Finney. Address correspondence to Mrs. Finney at Box 466, Department of Medicine, University of Virginia, Charlottesville VA 22908.

Presented 3-2-83.

The patient consulted his local physician twice during the first week of illness. He was given diphenoxylate with atropine and dicyclomine with phenobarbital, but he noted no improvement. During the second week of illness, he experienced occasional chills and feverishness. Over the two-week period of illness, he lost about ten pounds.

Four days before admission, he was seen in the emergency room of the University of Virginia Hospital. His vital signs were as follows: oral temperature 37.8°C, supine blood pressure 120/60 mm Hg with pulse 70/min, and blood pressure while standing 104/80 mm Hg with pulse 100/min. The abdominal examination was remarkable for active bowel sounds and diffuse tenderness without rebound; there was some localization of tenderness to the right lower quadrant. There was no palpable mass or organomegaly. The stool was guaiac-negative and there were a few white blood cells on the smear; a stool culture was subsequently negative for enteric bacterial pathogens, including *Clostridium difficile* culture and toxin assay. The total white blood count was 17,000/mm<sup>3</sup>, and electrolytes were normal. Abdominal films showed no free air or signs of obstruction; pancreatic calcifications and



gastrectomy staples were seen. The patient was given intravenous fluids and scheduled for elective admission to the hospital in four days.

On admission to the hospital, the patient denied having any gastrointestinal symptoms prior to this illness. He lived in rural central Virginia and had not traveled outside the state recently. A spring supplied water to his home. He had no history of exposure to a diarrheal illness, infected animals, or seafood. All other members of his family were well.

The patient had been an alcoholic for many years, consuming one quart of whiskey per day until 1978, when he stopped drinking. Seizures in 1977 were attributed to alcohol abuse. Insulin-dependent diabetes mellitus was diagnosed in 1963, and the patient took 20 units of NPH insulin each morning and evening. Peptic ulcer disease resulted in a partial gastrectomy in 1965. He was classified as permanently disabled in 1970 after sustaining a leg injury in a motor vehicle accident. He had smoked about half a pack of cigarettes every day for 43 years. There was a strong history of diabetes mellitus in his family. He had a history of rash to penicillin.

Physical examination on admission revealed an emaciated black man who appeared older than his stated age. His vital signs were as follows: oral temperature 37.6°C, supine blood pressure 126/70 mm Hg with pulse 76/min, blood pressure while seated 110/64 mm Hg with pulse 84/min, respirations 18/min and regular. He was 183 cm tall and weighed 53 kg. The skin showed decreased turgor and atrophic changes in the distal lower extremities. There was no palpable adenopathy. HEENT examination showed A-V nicking with a few hard exudates on the left. Cardiac examination disclosed a soft S<sub>4</sub>, no murmurs, and strong peripheral pulses. Abdominal examination revealed active bowel sounds and a visible, easily palpable, 4 × 7 cm fixed mass in the right lower quadrant just to the right of the midline. This area was markedly tender to palpation and there was rebound tenderness over the mass; the remainder of the abdomen was not tender. Liver span was 7 cm and the spleen tip was not palpable. Rectal examination revealed a slightly enlarged, smooth prostate and soft, guaiac-negative stool. There was evidence of degenerative joint disease in both knees.

The laboratory values on admission were as follows: hematocrit 34%, hemoglobin 11.2 g/dl, mean corpuscular volume 98.5, white blood count 14,700/mm<sup>3</sup> (6% bands, 87% granulocytes, 2% lymphocytes, 4% monocytes, 1% eosinophils) with toxic granulation of granulocytes. Platelets were

438,000/mm<sup>3</sup>, erythrocyte sedimentation rate 139 mm/hr, prothrombin time 11.8/11.2 sec, partial thromboplastin time 34.5 sec. Sodium was 136 mEq/l, chloride 100 mEq/l, potassium 4.0 mEq/l, bicarbonate 28 mEq/l. Glucose was 232 mg/dl, creatinine 1.1 mg/dl, BUN 12 mg/dl, total bilirubin 0.6 mg/dl, uric acid 3.5 mg/dl, calcium 7.7 mg/dl, phosphate 3.1 mg/dl, magnesium 2.0 mg/dl. Total protein was 6.4 g/dl and albumin 2.4 g/dl. Liver enzymes showed SGPT 11 IU/l, SGOT 9 IU/l, and alkaline phosphatase 142 IU/l; amylase was 21 IU/l. Urinalysis was clear, with a specific gravity of 1.025, pH 6, trace protein, glucose 1%, and 1-5 WBC/hpf. Chest X-ray showed a few granulomas. Stool examination was negative for ova and parasites; however, clumps of granulocytes were noted.

Upon admission to the hospital, the patient was started on intravenous fluids and placed on regular insulin on a prn schedule. Proctoscopy performed the morning after admission showed a large amount of semisolid yellow stool and erythematous but nonfriable mucosa. At 15 cm there was a mass effect pushing from the right. There was no involvement of the colonic mucosa. Stool obtained at 18 cm showed no white cells on wet prep.

That evening, the patient's temperature rose to 38.6°C, but there was no change in the abdominal findings. The diarrhea had diminished with institution of a clear liquid diet. He was examined by a surgeon and an abdominal sonogram was done. The sonogram showed multiple fluid-filled loops of bowel and an 8-cm complex mass with both cystic and solid components in the right lower quadrant. The radiologist was unable to discern the precise nature of the mass or the structure from which it arose.

An operation was performed on the second hospital day.

DR. ANTHONY BUSCHI: The abdominal film obtained at the time of his emergency room visit four days before admission (Fig. 1) shows heavy calcification within the entire pancreatic bed. The sigmoid colon appears to deviate from right to left, toward the left margin of the pelvis. Dr. Hook asked me earlier whether the few particles of air on the right side of the pelvis could represent extraluminal air. They could, but serial X-rays would be required to confirm this—if they did not change, they would most likely be intraluminal.

The abdominal sonogram obtained on the first hospital day (Fig. 2) shows multiple collapsed or fluid-filled loops of gut in the right flank. There is no evidence of renal obstruction and no evidence of any abnormality of the aorta or vena cava. A mass, mostly cystic but with some solid components, is

visible within the right lower quadrant. One cannot specify what the fluid is.

### DISCUSSION

DR. HOOK: Was an appendectomy performed at the time of gastrectomy in 1965?

DR. WILLIAMS: No.

DR. HOOK: I come clutching a copy of Cope's *Early Diagnosis of the Acute Abdomen*, which must be one of the most popular books in the medical library—the 12 copies in the stacks are checked out most of the time. However, I do not believe it helped me very much with this case.

There are some features of this patient's medical history that may well have contributed significantly to his present illness, and I will first comment on these.

As the abdominal film demonstrated, he had extensive calcifications in the pancreas. With rare exceptions, the finding of pancreatic calcification to such an extent is more or less diagnostic of chronic pancreatitis. Most cases of chronic pancreatitis occur in chronic alcoholics, and this patient had a long history of alcohol abuse. Although chronic pancreatitis is usually marked by recurrent episodes of abdominal pain associated with the acute inflammatory reactions, about 5% of chronic cases develop without significant pain.

There is a high incidence of pancreatic insufficiency resulting from chronic pancreatitis; the destructive process in the pancreas produces endocrine as well as exocrine deficiencies in the body which are frequently manifested as malabsorption and diabetes. Inadequate delivery into the intestinal tract of lipases and proteases produced by the pancreas results in abnormal digestion, with asymptomatic manifestations of malabsorption appearing almost immediately and symptoms of malabsorption developing once 85%-90% of the secretory reserve of the pancreas has been lost. The usual clinical manifestation is steatorrhea; carbohydrate malabsorption does not develop because salivary amylases are still present. This patient's pancreatic insufficiency, while not distinctly symptomatic, must have contributed to his recent weight loss—he weighed only 117 pounds on admission—and to his emaciated appearance.

Chronic pancreatitis is also related to development of diabetes: Two-thirds of the patients with chronic pancreatitis exhibit glucose intolerance and one-half of them are overtly diabetic. It is possible that pancreatitis brought about the diabetes in this patient, but genetic factors probably played a role,

too, for he has a strong family history of diabetes.

Patients with chronic pancreatitis can also develop pancreatic pseudocysts. These encapsulated collections of pancreatic secretions are seen in about 5%-10% of cases of chronic pancreatitis. Usually they form in the lesser sac, adjacent to the pancreas, but sometimes they burrow retroperitoneally and extend into the pelvis or mediastinum.

Major gastric surgery, including gastrectomy and gastroenterostomy, with or without vagotomy, enhances one's susceptibility to severe intestinal infections, such as those caused by *Salmonella* species, *Vibrio cholerae* and a number of other enteric pathogens. This patient had a subtotal gastrectomy 18 years ago, and the prominence of his diarrheal illness should prompt us to consider his vulnerability to intestinal infections. Normal gastric acidity, with a pH as low as 2, is rapidly bactericidal for many enteric pathogens and so constitutes a natural barrier protecting the gastrointestinal tract. Anything which alters the nature of this barrier, such as surgery or even the administration of antacids, and thus permits more viable organisms to pass into the lower gastrointestinal tract where disease can develop will enhance susceptibility to infection and, on occasion, to severe illness. I am most familiar with this phenomenon in connection with *Salmonella* infections. A survey of, say, 100 cases of *Salmonella* gastroenteritis will disclose a certain number of patients with prior gastrectomy; in them, the disease tends to be particularly severe.

In addition, patients who have had surgery for ulcers of various types exhibit an increased propensity to develop chronic gastrointestinal disturbances and diarrhea. This is in part true because hypertonic material enters the small bowel much more rapidly than it would under normal conditions.

The patient's present illness commenced with sharp pain in the right lower quadrant. He then developed rather striking diarrhea and a tender right lower quadrant mass. The mass was probably evolving for quite some time, perhaps from the onset of his illness. The sonogram showed that the mass was cystic and fluid-filled. It bore all the characteristics of an inflammatory mass: It was sharply localized, with marked direct and rebound tenderness, and it was associated with fever and leukocytosis.

I have concluded that the mass was a right lower quadrant abscess which formed subsequent to perforation of one of the principal occupants of the right iliac fossa, either the terminal ileum, the cecum, or the appendix. There is nothing to suggest



that the mass was vascular. It did not appear to be solid and so was probably not a neoplasm. Most infectious processes that develop in lymph nodes can become suppurative to this extent, but this is an unlikely prospect. Viral or bacterial intestinal infections can produce lymphadenitis without suppuration but with a focus of tenderness in the right lower quadrant and mimic appendicitis to the extent that an appendectomy is performed. All things considered, this was an abscess.

A great many pathologic processes can result in formation of an inflammatory mass in the right lower quadrant. Clearly, acute appendicitis is the most common of these, and, as Cope's book indicates, it should rank high in the differential diagnosis of any acute surgical abdomen.<sup>1</sup> From a statistical standpoint alone, one must look carefully at the possibility of appendicitis in this patient, even in the face of some atypical symptoms, because acute appendicitis accounts for about half of all acute surgical emergencies. Although the amount of diarrhea this patient had is unusually great and his abdominal pain did not begin in the umbilical or epigastric region, he did exhibit the classic march of symptoms of appendicitis: abdominal pain followed by nausea and anorexia, right lower quadrant tenderness, fever and leukocytosis. We would have to postulate rupture of the appendix in this case, something which occurs in about 5%-10% of cases. The rupture would have occurred early in the course of his illness, with subsequent development of an extraintestinal abscess. When proctoscopy was performed, the mass was found to be pressing



Fig. 1: Abdominal film shows pancreatic calcification and deviation of sigmoid colon from right to left.

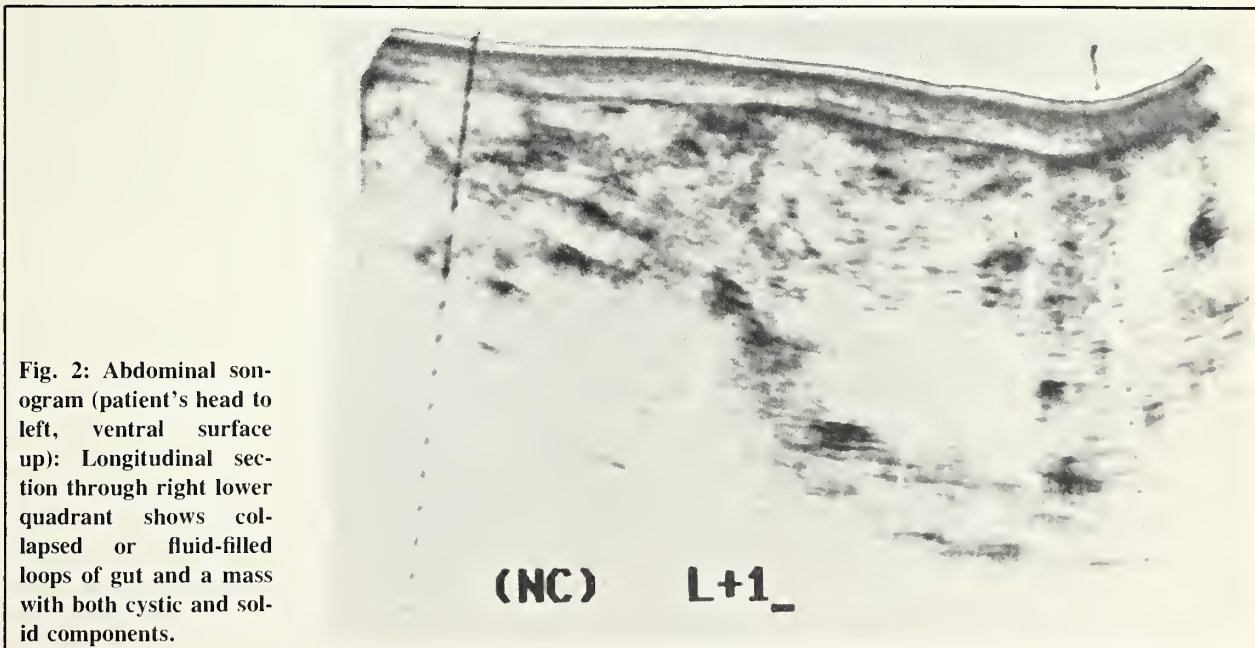


Fig. 2: Abdominal sonogram (patient's head to left, ventral surface up): Longitudinal section through right lower quadrant shows collapsed or fluid-filled loops of gut and a mass with both cystic and solid components.

on the rectosigmoid. Any inflammatory mass adjacent to the gut can produce inflammation of the gut wall, result in a few leukocytes in the stool, and contribute to development of diarrhea and tenesmus. The classic concept of the pathogenesis of acute appendicitis involves obstruction and distention, ischemia, bacterial invasion, gangrene, and rupture. An obstructive lesion—a fecalith or calculus, parasites, or an adenocarcinoma or carcinoid tumor—is found in about half of the cases.

I should add that acute diarrheal disease such as that seen in this patient may in some instances be related to or mimic acute appendicitis. Bacterial or viral enteritis due to *Salmonella*, enterotoxigenic *Escherichia coli*, *Vibrio parahemolyticus* or other pathogens may produce manifestations of appendicitis without any specific involvement of the appendix. In some cases, particularly with *Salmonella* infections, the organisms actually invade the appendix, and a certain proportion of cases of acute *Salmonella* gastroenteritis or enteritis are associated with pathologically visible appendicitis. Finally, there are other pathogens, notably *Yersinia* species, that characteristically involve the distal ileum and cause an illness that in a striking way resembles and may be confused with appendicitis. Five or six years ago, there was an outbreak of *Yersinia enterocolitica* infection in Buffalo, New York, in which the disease so mimicked appendicitis that there was an epidemic of appendectomies.<sup>2</sup>

A second cause of perforation of the bowel in the right lower quadrant is a cecal malignancy, most probably an adenocarcinoma. Carcinoma of the colon is, after skin cancer, the most common malignant disease in the United States. It occurs more often in men than in women and the majority of cases develops in the sixth or seventh decade of life. Seventy percent of the cases involve the rectum or sigmoid; the other 30% are distributed around the colon, with about 7% each in the cecum, ascending, transverse, and descending colon. Carcinoma of the colon causes perforation in two ways: it can actually invade the bowel wall or it can produce obstruction. The part of the large bowel that may rupture in a distal obstruction is the cecum. Alternatively, a neoplasm might cause obstruction at the appendix, producing appendicitis and rupture. Note that this patient's alkaline phosphatase was mildly elevated, and this raises the possibility of liver metastases.

Another diagnosis to consider is diverticulitis, which occurs in about 30% of persons over 60 years of age. It is most commonly found in the sigmoid and usually produces difficulty in the left lower

quadrant rather than on the right. Diverticula consist of herniations of mucosa or submucosa between bundles of circular muscle on the mesenteric side of the large bowel. They may arise anywhere in the colon and may rupture, producing pericolic abscesses, fistulae and pelvic masses mimicking carcinomas. Meckel's diverticulum, a common congenital anomaly, can give rise to a clinical picture closely resembling that of appendicitis, cause recurrent pain and bleeding, and result in intussusception. In other words, Meckel's diverticulum can produce precisely the right lower quadrant picture that we see in this patient.

The cecum is quite mobile and there are about 100 reported cases of volvulus of the cecum. Volvulus can interfere with the vascular supply to the affected region and lead to ischemia and perforation. Intussusception is another mechanical problem that may occur in the right lower quadrant.

The differential diagnosis of a right lower quadrant process must include Crohn's disease, which classically involves the terminal ileum and produces symptoms of low-grade obstruction, weight loss and abdominal pain; often there is a right lower quadrant mass and fever. Crohn's disease is sometimes diagnosed at surgery when a patient has been suspected of having acute appendicitis. This patient could in fact have Crohn's disease, but it would be unusual for Crohn's disease over such a short course to cause the abscess that I think this patient had.

Among the chronic infectious processes that can affect the terminal ileum, the cecum, or the appendix are tuberculosis and actinomycosis. Tuberculosis involves the terminal ileum and is frequently associated with a mass and so may be mistaken for Crohn's disease. Gastrectomy is thought to enhance one's susceptibility to pulmonary tuberculosis and it used to be said that ileal tuberculosis could occur only in the presence of pulmonary tuberculosis. That concept has changed in recent years, I believe, and there are a number of reports of ileal tuberculosis developing in patients who have no overt pulmonary tuberculosis disease. The time course of illness in this patient is very brief for tuberculosis and is a strong argument against this diagnosis. The same is true for ileal actinomycosis, which usually develops subsequent to cervicofacial, thoracic, or abdominal actinomycosis. The various forms of actinomycosis are usually associated with trauma or aspiration of foreign material. The abdominal type, which characteristically occurs in the ileocecal junction and may present with fistula and abscess formation, usually follows penetrating trau-



ma and may occur after appendicitis. Amebiasis is another infection to consider, for amebiasis of the colon can cause formation of amebomas. These inflammatory masses typically develop in the cecum and produce a filling defect that on a radiograph mimics carcinoma. The principal point against this diagnosis is that the patient's stools were negative for ova and parasites.

Were this patient seen in Africa, Asia or north-eastern Brazil—that is, in any part of the world where enteric fever is observed frequently—one would have to consider the possibility that he suffered a rupture of hyperplastic and necrotic Peyer's patches in the terminal ileum. Enteric fever, typically caused by *Salmonella typhi* but sometimes by other *Salmonella* species, is a prolonged febrile illness characterized by splenomegaly, leukopenia and lymphoid hyperplasia. Perforation of the terminal ileum occurs in about 1% of cases. In the United States, we see no more than a few hundred cases of enteric fever each year, and most of them are related to travel outside the country. That this patient had no recent history of foreign travel and negative stool cultures would be two strong arguments against this diagnosis.

I conclude, then, that this man, with his history of chronic alcoholism, gastric surgery, pancreatic insufficiency with clinically apparent malabsorption, and diabetes mellitus, perforated his intestinal tract in the right iliac fossa, in the appendix, cecum or terminal ileum. From a statistical standpoint, the most likely diagnosis is acute appendicitis with rupture. However, as a second possibility, I submit that a neoplasm such as an adenocarcinoma may have either caused the appendicitis or was itself responsible for the perforation. A third possibility is that the entire clinical picture was due to an infectious process such as that produced by *Yersinia enterocolitica*; the organism may not have been detected on the first culture.

### PATHOLOGY

DR. SCHWARTZMAN: The operation performed on the second hospital day was a partial ileocolostomy involving 60 cm of the terminal ileum and 30 cm of the cecum and ascending colon with the mesentery and appendix. The mesentery was firm and thickened and contained multiple enlarged lymph nodes, many of which were centrally necrotic and hemorrhagic. In addition, there was a fibrinous exudate matting the nodes together on the surface of the mesentery and matting the mesentery to the serosa of the bowel. The ileal mucosa appeared to be

inflamed and there were multiple small ulcerations with edema near the ileocecal valve. The mucosal pattern, however, was largely preserved and there was no gross evidence of perforation or tumor. The cecum and the colon were edematous and had small mucosal erosions. The serosal surface of the appendix appeared to be inflamed. Thus the principal features of the surgical specimen on gross inspection were necrosis and hemorrhage of the mesenteric lymph nodes. This indicated that an acute bacterial process, rather than a chronic process such as Crohn's disease or ulcerative colitis, was responsible for the patient's illness.

Examination of microscopic sections revealed ulcerations in the terminal ileum. The mucosa, while preserved, was focally ulcerated. Inflammation was present in all layers of the bowel. There was a marked acute inflammatory infiltrate penetrating through the muscularis and into the mesentery and serosa of the bowel. Lymphocytes and plasma cells were scattered here, but most of the cells were polymorphonuclear leukocytes.

The microscopic section of a lymph node revealed that, while the basic architecture of the node was largely intact, its center was entirely necrotic. One could see an abscess forming, and a highpower view of the abscess showed that it was filled with inflammatory cells as well as with macrophages, lymphocytes and plasma cells.

No granulomas were found in any of the lymph nodes, an important negative finding suggestive of a bacterial etiology. Indeed, a surgical specimen grew a pure culture of a gram-negative bacillus subsequently identified as *Yersinia enterocolitica*. This organism can be grown on standard media used for screening stool isolates but may be mistaken for *E coli* or *Proteus* and discarded. There is no good diagnostic serologic test for *Yersinia* due to cross-reactivity with other bacterial antigens. Serology is therefore reserved for use in epidemiologic investigations.

DR. HOOK: This is an extremely interesting and instructive case. We have learned quite a bit in recent years about *Yersinia* and the infections it causes. We might ask Dr. Richard Guerrant, head of our Division of Geographic Medicine, to comment briefly on *Yersinia* and this patient's illness.

DR. RICHARD L. GUERRANT: When this man was admitted to the medical service, we felt that the most likely diagnosis was either appendicitis with rupture or a carcinoma. Dr. Hook quite accurately suggested, too, that he could have had a classic enteric fever-like illness. I was curious to know whether there actually was involvement of Peyer's

patches, because in some models of *Yersinia enterocolitica*, as with *Salmonella typhi*, infection is initiated there.

Studying this patient's course in retrospect, I would say that it was quite typical of the few *Yersinia* cases we see in this country. His illness began with watery diarrhea which became inflammatory over a two-week period. The presence or absence of a few leukocytes in the stool may not be all that specific in someone who obviously has an inflammatory process in the right lower quadrant, but the recent association of *Yersinia enterocolitica* with an enterotoxin makes one wonder whether the watery diarrhea was an early phase of an enterotoxic diarrhea which then developed into an enteric fever-like illness.

This man had only the spring water at his home as a known possible source of infection.

DR. HOOK: It is quite likely that his gastrectomy altered his susceptibility to *Yersinia*.

DR. WILLIAMS: I have some followup information on this patient. His postoperative hospital course, although prolonged, was fairly uneventful. He received cefoxitin and tobramycin after surgery, and the former was continued when antibiotic sensitivity testing showed the *Yersinia* to be susceptible to cefoxitin. Other antibiotics which may be used in *Yersinia* septicemia and intraabdominal abscess include gentamicin and other aminoglycosides, cefamandole, and chloramphenicol.<sup>3</sup> A followup stool culture done for this patient one month after discharge was negative. He was readmitted to the hospital three months later for closure of his ileostomy. He is doing well now.

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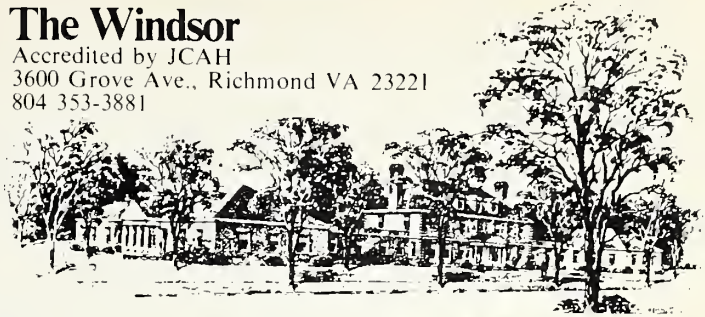




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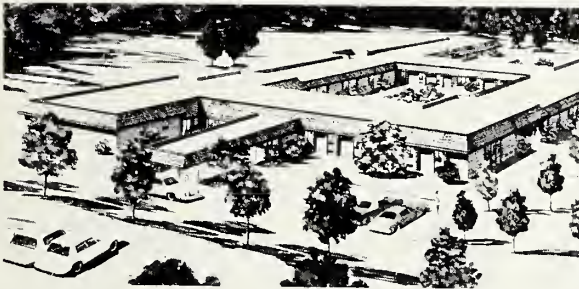
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## Virginia's Board of Medicine

is 100 years old this year, and in this picture are gathered members of the licensing body in its centennial year; they represent medicine, chiropractic, clinical psychology, osteopathy and podiatry. In the front row, from left to right, are Edwin T. McNamee, Jr., MD, Stuart, the Board's president; Barbara A. Mella, MD, Fairfax, the first woman to sit on the Board since its founding in 1884; and George J. Carroll, MD, Suffolk, for more than a decade the Board's secretary-treasurer. In the second row, from left, are George Edward Calvert, MD, Lynchburg; Charles N. Van Horn,

MD, Norfolk; Joshua P. Sutherland, MD, Grundy; and James B. Blayton, MD, Williamsburg. Third row, from left, Abraham A. Coster, DPM, Alexandria; Gerald J. Bechamps, MD, Winchester; and Franklin J. Pepper, MD, Alexandria. Fourth row, James W. Walker, DC, Charlottesville, Harold A. Blood, DO, Alexandria; and Wayne E. Engel, PhD, Harrisonburg. Behind them all is Mrs. Eugenia K. Dorson, for twelve years the Board's executive secretary. Not present when this picture was made was the Board's vice president, Edwin L. Kendig, Jr., MD, whose editorial appears at right.



# VIRGINIA MEDICAL

## The Regulation of Virginia Medicine

**D**URING its annual session in November, the House of Delegates of The Medical Society of Virginia voted unanimously to commit the Society to opposing the transfer of Virginia's health regulatory boards to the State Commerce Department. I emphatically agree with that position.

The suggestion for such a change originated in the Virginia Joint Legislative Audit and Review Commission. The commission's staff report may have come across your desk, as it did mine. It will no doubt lead to the introduction of legislation at the 1984 General Assembly beginning this month. The recommendation that the health regulatory boards be merged with (placed under the control of) the Department of Commerce presents a disturbing move against the medical profession.

The State Board of Medicine was earlier under the aegis of the Department of Commerce. However, at the combined request of the State Boards of Medicine, Pharmacy, Nursing and Dentistry, legislation creating a Health Regulatory Commission was introduced and passed into law (1977). In initiating this proposal for a Health Regulatory Commission, the goal of the health boards was not that of greater regulation of their activities but rather the provision of an improved, as well as a cooperative, approach to the individual, and sometimes common, problems of these related boards.

As the result of a recent recommendation of an internal study carried out by the Management Analysis and Systems Development Department, medi-

cal care in Virginia (through the health regulatory boards) will be controlled more and more by non-physicians and with the expertise of the physicians being not so gradually relegated to a minor level of influence. The director of the health regulatory boards is a man of integrity and capable, too, but he is not a health professional, and more and more authority is being assigned to this office.

The Health Regulatory Commission should continue to be maintained separately from the Department of Commerce, a body which deals with a large group of completely unrelated agencies. However, this commission should be reconstituted to be composed of the same number of members (14), including a representative of the original four boards (medicine, pharmacy, nursing and dentistry), as well as one from veterinary medicine, optometry, behavioral sciences and seven public members. The position of full-time director of the health regulatory boards should be programmed to be that of a coordinator and business manager, nothing more. The other board currently under the Health Regulatory Commission, i.e., the Board of Funeral Directors, should be transferred to the Department of Commerce.

Just as the legal profession retains the right to regulate its own affairs through the State Bar Association, those of us in the medical profession feel strongly that medical affairs should be regulated by physicians.

EDWIN L. KENDIG, JR., MD

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# PRESIDENTIAL ADDRESS

## **“Is it worth it? Who will pay for it? Who will control it?”**

Mr. Speaker, Dr. Rogers, honored guests, colleagues, ladies and gentlemen.

It has been an interesting and challenging year.

Let me, first, introduce the members of my family who are here—my wife, Ann, and my daughters, Carol and Penny. Three of my children, Mark, Kay and Jeff, could not be here because of commitments at work.

Few can come to this position in life without the help and support of a good wife and family. All of these have been understanding and supportive of a husband and father, who has all too often been away—usually at the hospital way past their bedtime—for more years than he would like to recount. I think they know their understanding has been appreciated.

It has been a privilege and an honor to serve in the highest position my colleagues and this House can bestow—the presidency of The Medical Society of Virginia. Nothing will ever equal this year. I thank all of you.

Also, I would like to remember and honor at this time a man who not only taught me surgery but also taught me that patients and their problems come first, the one must always be willing to go back to that hospital and that bedside as many times as necessary to make sure that all possible is being done—the late Dr. John M. Emmett. Some of you will remember that Dr. Emmett was President of this Society in 1943. He was a great teacher and a compassionate human being.

I also wish to thank my lone partner, Dr. John C. Wirth, Jr., who agreed to change days and weekend call when necessary to allow me to attend the many functions of this Society.

Your Council and the committees of this Society have had a busy year. Their reports are in your

packet and I commend them to you. There are several committees which have necessarily been more active than others, and, I believe, deserve special mention at this time.

The Insurance Committee, chaired by Dr. Alvin Conner, has done an outstanding job in an increasingly difficult and complex area.

The Legislative Committee, under Dr. Glen Updike, continues to provide expertise and leadership in the field of legislative action.

The Vanguard Committee, under Dr. Hayden Hollingsworth, has spent many hours considering the many federal bills and policies and has charted a course they feel we should follow.

The Physicians Health and Effectiveness Committee, so ably chaired by Dr. William Barney, has put into place a solid plan which will reap benefits far into the future.

The Membership Committee, under the guidance of Dr. Thomas Hulvey, and the Bylaws Committee, chaired by Dr. Charlie Caravati, have solved, I trust, the technical problems of resident membership in this Society.

The Education Committee, chaired by Dr. Gene Temple, with the invaluable assistance of Dr. Kinloch Nelson, has sponsored several risk management courses for all of us, in addition to their many other responsibilities.

The PSRO Liason Committee has been phased out and the MSVRO phased in, as directed by this house last year. Dr. Robert Morton has become the chairman of this planning group, and it appears all the necessary ground work has been done to be ready to negotiate with the Department of Health and Human Services for peer review.

As you have heard this afternoon, the Auxiliary, led this year by its president, Mrs. Elaine Weitzman, continues to support us and the goals of this Society in so many ways. I will personally give them our thanks at their luncheon meeting tomorrow.

Presented before the House of Delegates of The Medical Society of Virginia on November 3, 1983, at the Homestead, Hot Springs, Virginia.



I could not overlook the fine work done by Dr. Mike Puzak and his Finance Committee, whose report you will hear later today.

In addition, this Society and VAMPAC continue to work closely to advance medicine's best interests and I would urge at this time that all of you, who have not done so, join with us to make this organization even more effective. Dr. Percy Wootton, chairman of VAMPAC, and our special guest, Dr. Fred Rainey, chairman of AMPAC, will speak to us concerning this vital organization later today.

I would like to thank the Virginia delegation to the AMA, led by Dr. Puzak, for including the officers of this Society in their deliberations in Chicago and Miami Beach this past year, and commend them for their conscientious attention to the issues. We are represented well at the highest level of organized medicine.

Through all of these committees and their work runs the fine thread of our Executive Vice President, Jim Moore, and his staff, and the legal guidance of Allen Goolsby and his associates.

Certainly, one of the highlights of the year was our participation in the Summit Conference of Industrialized Nations in Williamsburg. On behalf of this Society, 24 physicians and 13 nurses volunteered their time and knowledge to assist the State Department in hosting this conference. The amount of good-will generated was immeasurable and, although no serious emergencies occurred, we were ready for anything, including cardiac arrest. President Reagan has kindly expressed his appreciation to this Society and to all of you.

Another highlight was the Congressional Luncheon with our representatives in Washington. A full day was planned by Ed Debolt and his staff, and Council had the unique opportunity to talk with both senators and nine House members in a most informal way.

Mr. Speaker, in the interest of time, I would like to simply list several recommendations which I respectfully desire this House to consider:

1) that this Society support the concept that all patients have the privilege of assigning their rights to payments from all third party carriers, including the federal government;

2) that the President-Elect be compensated in the amount of \$3,000.00 per year;

3) that the Speaker of the House of the MSV be a part of the Virginia delegation to AMA meetings;

4) that recommendation Number One of the Legislative Committee and the resolution of the Norfolk Academy on informed consent for breast cancer patients be strongly supported;

5) that this House consider adding more specialty society sections;

6) that consideration be given to the establishment of a "financial services" department for our members;

7) that Report D of the Council on Medical Service of the AMA be strongly supported;

8) that Jim Moore and his staff and Allen Goolsby and his associates be commended for a job well done;

9) that Dr. Edwin Kendig, Jr., Mrs. Ann Gray and the Editorial Board be commended for the continued excellence of our publication—VIRGINIA MEDICAL;

10) that Ed Debolt, Carole Cones and their staff be recognized for the strong support they give to many of our activities.

Now allow me to speak briefly about what I believe to be the most serious and important problem facing medicine today—the cost of medical care. Any discussion of this topic is most important because it impacts upon the quality of care, the delivery of care, and upon the fundamental concepts of doctor-patient relationships.

Politicians and government delight in promising everything they can—usually near election time—but when the cost of one of their programs gets out of hand, they look around for someone else to take the blame.

In 1965—rather than help only the needy—Congress chose to pass legislation that covered the cost of medical care for *all* those persons over age 65—regardless of need. They were told by the leaders of this profession that the cost would be tremendous and that eventually no one would be able to pay the bill. We treated some of those same patients in 1964 free of charge and would do so again if that becomes necessary. But "free" care at that time was a stigma.

Now, rather than admit that they can't keep their promises, government seeks once again to blame the medical profession and force us to accept less and less of our just fee to help *them* fulfill *their* promises.

As I travel around to the state meetings of other societies, the topic of medical costs is discussed at length. I have had the opportunity to hear Dr. Edward Annis speak several times. What a spokesman for the medical profession! He always asks the intriguing question, "Why doesn't government pay their medical bills?" They pay all their other bills. They pay for tanks, planes, ships. When aircraft carriers are built in Newport News, everybody gets paid—the architects get paid, the contractors, the

pipefitters, the welders—they all get paid—they even get paid for cost overruns. And they don't get 80% of some number—they get 100%. The interest on the national debt also gets paid—and paid promptly. Can you imagine what would happen if government did not pay the interest on the national debt—even for just one day? Now, that would be a crisis! But with medical bills, they somehow seem to be saying "We can't afford it."

Can't afford the best medical care in the world? Can't afford to keep patients alive on dialysis who would otherwise die? Can't afford intensive care for the elderly?

Let's examine a few facts. We hear that the medical care costs have risen from 9% to 10.5% of the gross national product—at a much faster rate than inflation. Of course medical costs have risen. Consider new technology—CT scans have revolutionized the field of neurology and neurosurgery. Neonatal ICUs, coronary care units and imaging now make it possible to save lives and make people—many of them elderly—live longer, with a better quality of life—not possible ten years ago. Transplant surgery is exploding, including liver transplants for infants, which we have heard so much about lately.

Costly research is necessary for these advances, and costly surgical care is also required. There is still no "free lunch" in America. Do we wish to stop research? Do we want to stop the production of better and more accurate diagnostic tools? I hear no one advocating that we stop liver transplants for children. And I have never heard a relative outside the intensive care unit say he wanted anything but the best of care for his grandmother.

Let's consider, again, that figure of 10.5% of the GNP.

Is this too high for medical care? As Dr. Annis points out, 17½% of GNP goes for sports and recreation. Shall we try to limit this? How about putting a limit on the salary of every sports star, and reduce the price of tickets? I don't think we would get very far.

Twenty percent of the GNP is spent on transportation. Shall we stop building roads and rapid transit systems?

Even the statistics on percent rise in health costs are misleading. The cost of CT scanning or neonatal intensive care, for instance, is "new care" and could not be purchased five years ago—by anyone—at any price. Comparing the rise in cost of items of this kind, and there are many, to say, the rise in the cost of food, clothes or automobiles, is really unfair.

It is time that we, as a profession, stopped feeling guilty or apologizing for rising health care costs.

We have the best medical system in the world. People are living longer with a better quality of life. Infant mortality is coming down each year. The death rate in this country has dropped each year for ten years. More people are receiving better care than ever before.

This is not to say that medical costs cannot be reduced in some areas. We can admit fewer patients and do more procedures in an outpatient setting. We can order fewer lab and X-ray studies, although we have to be careful here to preserve quality. We can make sure patients go home from the hospital as soon as they safely can. We can hold physicians charges at reasonable levels. We are beginning to do these things, but all these efforts and all the efforts of others in the health care field, including DRGs, will have a relatively small impact, and will likely *not* keep the cost of total health care from rising. This cost will assume a slowly increasing share of the GNP—primarily because of ever-increasing technology and the rightful demand of the public to have the best care possible. I ask you—what is more important than health?

To deny care, delay care, or give less than the best care represent the only real ways to cut costs—and none of these is acceptable in this country. Incidentally, I hear that in England there are 800,000 people on the waiting list for elective surgery, and they wait anywhere from two to six years.

In this atmosphere of public debate on health cost, there have arisen many proposals for cost containment and pro-competition, which have been put forward to cure all of our problems, and to somehow *magically* reduce the cost of getting a patient well again. Some of these are well-intentioned and others not. Some would seem to be much more interested in control of prices and methods of health care delivery than with patient care or the quality of that care.

The crisis, if there is one, is not the cost of medical care, but rather: Is it worth it? Who will pay for it? Who will control it?

If the private practice of medicine, as we know it, is to continue, we must actively defend and promote this profession. We must—each of us—become informed and willing to talk to anyone who will listen—our patients, civic groups, friends, congressmen—anyone—and tell the positive story of medicine at every opportunity. Our greatest allies are our patients. It is estimated that we see 2,000,000 a day across the country.

I believe that the public, once informed, will



understand the why of health costs, and will agree that the results are worth the price.

We have an obligation to those who have gone before and those who will come after, to sustain those principles which have made this profession so respected and so responsive to patients' needs over the years. We must preserve that special relationship that exists between a sick patient and his chosen doctor, that allows a mutual confidence and understanding to develop, that permeates all of medicine.

Anything that changes that special relationship between patient and doctor, I believe, runs the risk of undermining the best system of medical care yet devised in this world.

As you individually consider all the many plans and proposals by the government, lay groups and social planners, I hope you will weigh all the possible effects each proposal will have on patient choices and the free private practice of medicine and make your informed decisions according to your best judgment. The responsibility is great. Your decisions will determine the direction medicine will take from this point, and years into the future.

It is written that, in ancient times, when the Athenians decided that the freedom they wanted most was freedom from responsibility, they ceased to be free and were never free again.

Two hundred years ago, Thomas Paine wrote, "What we obtain too cheap, we esteem too lightly; 'tis dearth only that gives everything its value."

Is this profession not dear? Are the time-honored principles of medical practice not dear? Let it not be said of this generation that the issues were too complicated, the pressures too great, or the task too difficult.

Let us accept the challenge and fight for the principles in which we believe.

Just as this profession faces challenges, so does this country. In recent weeks this nation, and its ideals of freedom, have been challenged anew around the world. It is a good time to reaffirm our commitment to this great country, and to its institutions and to that American dream—

- That dream which says that if we work and strive we will prosper and improve our station in life, and that of our children.

- That dream which enabled a young surgeon from the valley of Virginia to suffer through the agony of the death of his beloved commanding general, yet go on to become the President of this Society, in 1881, and later President of The American Medical Association, and honored in his lifetime throughout the world.

- That dream which allows each of us the opportunity to be what we wish to be, to have freedom in our choices, to answer only to God and the laws of this land, limited only by the bounds of our imagination, the depth of our desire, and the strength of our convictions.

HAROLD L. WILLIAMS, MD

12511 Warwick Boulevard  
Newport News VA 23606

## Making Their Voices Heard: the Auxiliary

THE SONG, "Woman", begins, "I am woman, hear me roar in numbers too big to ignore." It was from that song that the Medical Society of Virginia Auxiliary took its theme for 1982-1983; and we did work hard to make our voices heard across the state.

Our membership was urged to focus its attention on current health concerns as they particularly affected women: alcohol and drug abuse, battered women and children, more cancer screening programs, hospice, and the problems of our senior citizens—what I have often referred to as the special health concerns of the '80s.

Our local projects included everything from "parenting" programs to a Nutrition Day for senior

citizens, from hospice to the endowment of a Women's Resource Center with a \$4,000.00 grant, from Tel-Med to the distribution of pamphlets on breast self-examination. There were blood pressure clinics, city-wide auxiliary-sponsored CPR courses, information booths at health fairs, health career scholarships, support of hospitals and emergency care facilities, and much, much more.

Involvement, too, was evident in the area of government relations. We organized "hot lines" within the Auxiliary and became more active in local politics and supportive of political action groups. Some of our members joined MSV councilors and members of the local medical societies in their joint meetings with state legislators; and we

avidly tried to keep abreast of medical issues aired in the *AMA News* and the AMA's "Legislative Report." We received an invitation to join the Society at its annual meeting to hear AmPAC speaker, Dr. Fred Rainey, and accepted with pleasure; and we look forward to a joint legislative workshop next year in Richmond.

One of the highlights of the Auxiliary year was our Legislative Day, which brought forth the largest turnout an event of this kind has ever had. Auxiliaries toured the Governor's mansion and Capitol Building in the morning and visited House and Senate committee hearings in the afternoon. Our luncheon speaker was Eva Teig, executive assistant to Lt. Gov. Richard J. Davis. The daughter of a physician, she has recently been appointed to head the State Department of Labor and Industry.

Our midyear board meeting was held in Virginia Beach, where we welcomed the AMA Auxiliary's AMA-ERF chairman, Mrs. Thomas Fields, as our special guest. Following this meeting a workshop was held in Richmond by the president-elect, Mrs. Rahmat Seif, for the 1983-1984 county presidents and their major committee chairmen; and it, too, was very successful. At that time I was enjoying a visit to Capitol Hill with the Society leadership and wives, including lunch with our congressmen and senators.

In the spring also AMA-ERF checks were presented to Virginia's three medical schools in the following amounts: University of Virginia, \$13,980; Medical College of Virginia, \$19,560.88; Eastern Virginia Medical School, \$12,659.12. The total, \$46,200.

At the AMA Auxiliary Convention in June, our eight delegates proudly displayed buttons with the #6 on them, representing six consecutive years of membership increase. Virginia received awards for the organization of the Suffolk and Chesapeake Auxiliaries and learned that we were among only 12 states maintaining or increasing membership this year.

The MSVA's Organ Donor Awareness Program, begun in April, received further impetus this summer when kits were mailed to county presidents and health projects chairmen, providing additional information on organ donation and lists of resource materials. This will be a continuing project.

We are now working on a second state project, the Impaired Physician Program and hope to establish a support group for the families of impaired physicians. An introductory seminar was conducted at the Homestead on November by Mrs. B. David Epstein of Florida, an AMA Auxiliary

director for the Southern Region. All physicians, their spouses and guests at the convention were invited to attend.

This year has been a most memorable one for me. It has provided me with a tremendous awareness and appreciation for the talents and capabilities of the women of our state and of the AMA Auxiliary and its components. There were visits with representatives of 21 of our auxiliaries, trips to Richmond to attend the Medical Society of Virginia Council meetings and the Governor's conferences on critical reevaluation, a trip to the Homestead for the planning of convention, and meetings galore.

Our bylaws were reprinted, our Handbook updated, and arrangements made for the annual meeting. The MSVA's keynote speaker this year was Mrs. Wayne C. Brady, President-Elect of the AMA Auxiliary. Other special guests included Mrs. William D. Hughes, president of the Southern Medical Association Auxiliary, and luncheon speaker, Dr. Harrison Rogers, Jr.

At our House of Delegates meeting, a vote was taken to amend the MSVA's bylaws to permit widows whose husbands were members in good standing of the AMA at the time of death to become eligible to be delegates to the AMA Auxiliary convention and passed by unanimous consent.

The 1982-83 year was to have come to an end for me at the annual luncheon following our House of Delegates meeting, but there were two encores: a meeting scheduled with the Surgery House Staff Wives Club at the Medical College of Virginia with guest speaker, Mrs. John G. Bates, President of the AMA Auxiliary; and the Virginia Women's Forum, sponsored by Miller and Rhoads and cosponsored for the past three years by the MSVA. It was a pleasure to have the special honor of being seated next to former Sen. Harry F. Byrd, Jr. at the luncheon following the Forum.

The support given the Auxiliary by The Medical Society of Virginia this year has been outstanding. We are especially appreciative of the efforts of Dr. Harold Williams, Mr. James Moore, Mr. Will Osborn, and Mrs. Donna Strawderman for all they have done to assist the Auxiliary and to enhance the working relationship between the two organizations. I thank one and all for the opportunity to serve as President of the MSVA and look forward to the continued growth and achievements of the Auxiliary under the leadership of my successors.

ELAINE WEITZMAN

2807 Acres Road  
Portsmouth VA 23703



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# VIRGINIA MEDICAL OBITUARY

## **Landon E. Stubbs, MD**

Dr. Landon Elwood Stubbs died February 20, 1983, at his home in Newport News. He was 94 years old.

A native of Gloucester County, Virginia, Dr. Stubbs was graduated from Randolph Macon College in 1910 and from Johns Hopkins University School of Medicine in 1914. He received training at the Washington, DC, Hospital Center and Peter Bent Brigham Hospital, Boston. He served in the Army Medical Corps during World War I and returned in 1918 to practice in Gloucester County. He moved to Newport News five years later, there to establish a practice that endured for more than 50 years.

In 1926, Dr. Stubbs was instrumental in transferring to The Medical Society of Virginia the deed to "Belroi", birthplace of Walter Reed, then owned by Dr. Stubbs' father, George Marvin Stubbs. Title to the historic site was again transferred in 1968 to the Association for the Preservation of Virginia Antiquities.

Dr. Stubbs came to membership in The Medical Society of Virginia over 63 years ago, through the Newport News Medical Society. He was also a member of the Southern Medical Association and the American Medical Association.

## **Daniel H. White, MD**

Dr. Daniel Henry White, neurosurgeon, Norfolk, died September 22, 1983, in a Virginia Beach Hospital. He was 43 years old.

A native of Hackensack, New Jersey, Dr. White was a graduate of Mount St. Mary's College in Emmittsburg, Maryland, and the University of Maryland School of Medicine. He trained at his alma mater and also at the Naval Regional Medical Center in Portsmouth, Virginia. In addition to his private practice, he was an assistant professor of

neurosurgery at Eastern Virginia Medical School.

His professional memberships included The Medical Society of Virginia, Norfolk Academy of Medicine, Seaboard Medical Association, Virginia Neurological Society, Congress of Neurological Surgeons and American Association of Neurological Surgeons.

## **Robert C. Wesley, MD**

Dr. Robert C. Wesley, Lynchburg general practitioner, died July 18, 1983, at the age of 71.

Dr. Wesley earned his medical degree in 1945 at Meharry Medical College, Nashville, Tennessee, and trained at the Harlem Hospital Center in New York. He then served for two years in the US Air Force before establishing his practice in Lynchburg.

Dr. Wesley came to membership in The Medical Society of Virginia 18 years ago through the Lynchburg Academy of Medicine. He was also a member of the American Academy of Family Physicians.

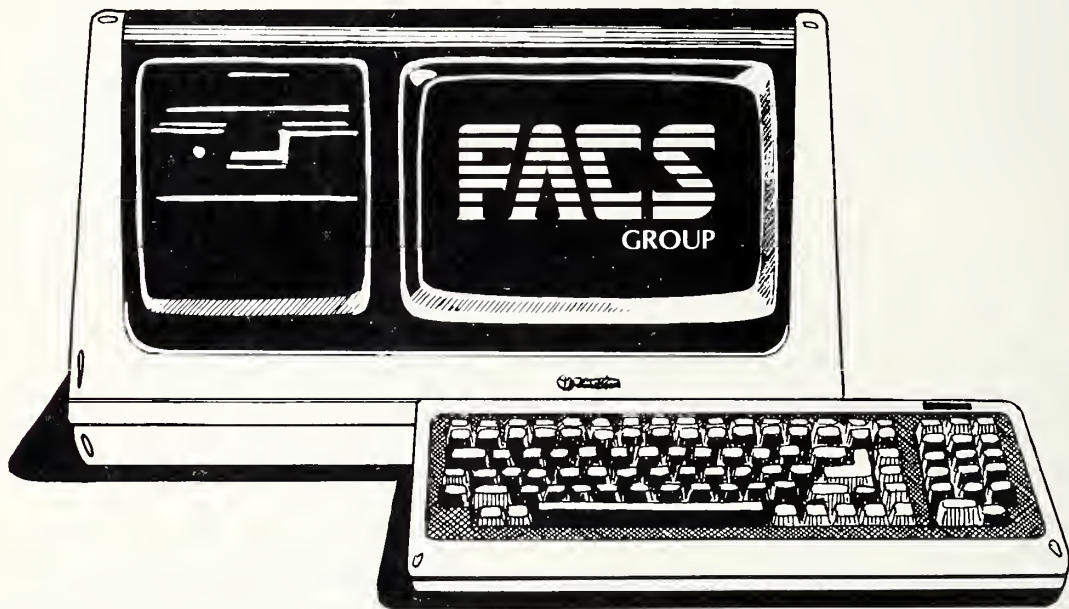
## **Harry Bernstein, MD**

Dr. Harry Bernstein, Farmville urologist, died November 10, 1983, at the Medical College of Virginia Hospital, Richmond. He was 75 years old and had retired from practice in 1980.

Dr. Bernstein received his medical degree from Temple University, Philadelphia, Class of 1928, and took training at Memorial Hospital, Philadelphia. He had been chief of surgery at St. Agnes Hospital in Philadelphia before moving to Farmville in 1972.

Dr. Bernstein was a member of the American Urological Association and American College of Surgeons. He came to membership in The Medical Society of Virginia through the Southside Virginia Medical Society.

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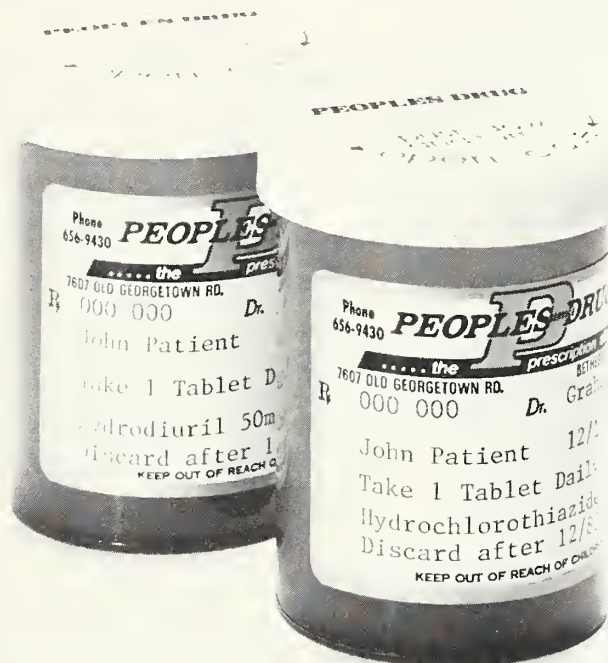
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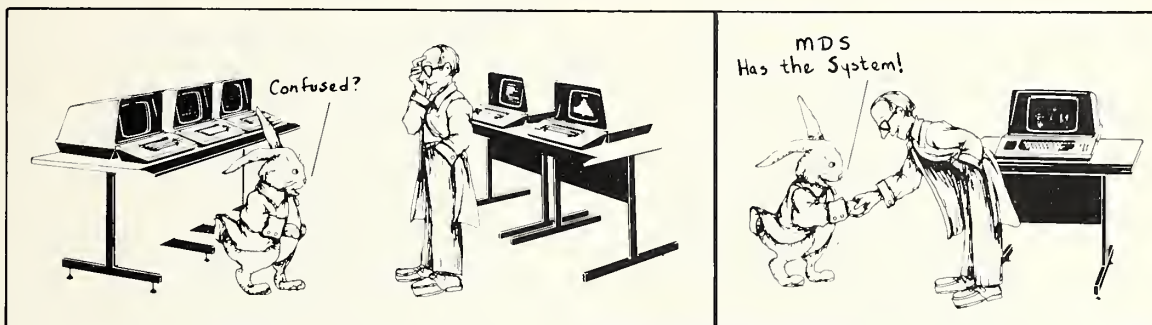
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# LETTERS

## Questions house call charge of home visit enterprise

I would like to congratulate the Editors on what I feel is marked improvement in the quality of VIRGINIA MEDICAL over the past year. I feel that the notes from the VIRGINIA MEDICAL News Bureau are quite entertaining and frequently edifying and are a nice complement to the scientific articles. Although I assume the readers of VIRGINIA MEDICAL are almost entirely physicians, I would suspect that some of the items are picked up by the lay press. It is for this reason that I would like to comment on a news item in the December issue.

My specific comment is focused on the discussion of "house calls"<sup>1</sup> in which one physician who is involved in a business enterprise of home visits makes the statement that her fee is \$64 per visit plus mileage for long trips and that this does not appear to her to be "out of line." In my opinion this charge is out of line and excessive. I am in a practice with three other internists and each of us makes several house calls during most weeks. Our charge is less than half that figure.

In my experience, house calls are seldom necessary and are usually an inefficient use of the physician's time. However, there are times when it is necessary to see someone at home. I hope that most of your physician-readers do not make such an extraordinary charge and that they agree with me that a fee of \$64 per visit is totally "out of line."

**William W. Martin, Jr., MD**

2201 Grove Avenue  
Richmond VA 23220

1. Old folks' needs rejuvenate a tradition. Va Med 110:682-683, 1983

**Dr. Cynthia W. Merrill, president of Physician Home Visits, Inc., responds:**

I agree with Dr. Martin that a charge of \$64 for a house call to one of my own patients would be excessive, but Physician Home Visits provides care for patients who have no doctors or whose doctors make no house calls. We do not see patients who are physically able to go to an office. Therefore, a

complete history and physical must be performed on all initial visits. These examinations, which may require an EKG, lab work or a catheterization, have averaged 1-1½ hours of the physician's time. Medicare allows \$40 of the fee, although for visiting nurses the Medicare reimbursement is \$54.

## Praise for review of Starr's history of American medicine

Dr. Charles E. Davis' review of Paul Starr's book, *The Social Transformation of American Medicine*, in the December 1983 issue of VIRGINIA MEDICAL<sup>1</sup> was excellent. I, too, found Starr's history of the evolution of American medicine to be fascinating and illuminating. Dr. Davis' comprehensive and thoughtful review of this book should stimulate every physician in Virginia to reflect upon how medicine got to its present situation and, knowing its history, how we might preserve its better aspects into the future. The review was superb.

**David K. Wiecking, MD**

Chief Medical Examiner,  
Department of Health  
9 North 14th Street, Richmond VA 23219

1. Davis CEjr: Medicine meets the marketplace. Va Med 110:688-690, 1983

## Stroke/salt text brings inquiry from Belgian researchers

My article on stroke mortality and salt ingestion in Eastern Virginia in the March 1983 issue of VIRGINIA MEDICAL<sup>1</sup> was of special interest to epidemiologists at the School of Public Health, University of Leuven, Leuven, Belgium. Dr. J. V. Joossens wrote to me that he and his staff have also been working on this topic for several years<sup>2-4</sup> and have linked salt intake not only to stroke mortality but also to stomach cancer mortality. He asked if I would reexamine my stroke data and determine if an association of stomach cancer mortality likewise existed for the same high-salt-intake population of the Northern Neck, Middle Peninsula and Eastern Shore of Virginia.

My statistical correlations did confirm a specific association of stomach cancer to stroke mortality, particularly for intracerebral hemorrhage. The association was strongest for non-whites living in rural counties as compared to urban areas. Gastric can-



cer mortality data followed stroke mortality proportionately from 1974-1978.

**George Moore, MD**

PO Box 226  
Warsaw VA 22572

1. Moore G: Stroke mortality and salt ingestion in Eastern Virginia. *Va Med* 110:159-165, 1983
2. Joossens JV, Geboers J: Nutrition and gastric cancer. *Nutrition Cancer* 2:250, 1981
3. Joossens JV, Geboers J: Salt and hypertension. *Prev Med* 12:53, 1983
4. Joossens JV, Geboers J: Cardiovascular diseases, cancer and nutrition. *Acta Cardiol* 38:1, 1983

### **Dr. Nelson's certification odyssey a source of education**

Thank you for ensuring my fame with the reprint about my beaver encounter in the November 1983 issue of *VIRGINIA MEDICAL*.<sup>1</sup>

The article in the same issue tracing Dr. Kinloch Nelson's odyssey through the certification process for his ambulatory surgery center<sup>2</sup> was one of the best I have read in any journal. One of the impressions I came away with was that no one has any firm basis for predicting whether any one form of inpatient or outpatient provision of services will cost more or less than any other method of doing the same thing. If articles such as this can educate us about the immensity of the sea of ignorance in which policy decisions are being made, presumably it could lead at least to less forcible and damaging decisions being made. In this context, the subtitle "Footprints on the Sands of Certified Need" seems especially appropriate.

**Lockhart B. McGuire, MD**

University of Virginia School of Medicine  
Charlottesville VA 22908

1. Shaw B: Just when you thought it was safe enough to go back into the creek . . . *Va Med* 110:619, 1983
2. Gray A: CON artist. *Va Med* 110:646-654, 1983

### **Longer film needed for taller people?**

It has been my observation over a period of years that people in the United States are growing taller. Frequently we are unable to get an entire long bone on a conventional X-ray film, even taking the film from corner to corner. Perhaps there should be a

design change in X-ray machines so that films can routinely be 20 inches long by 16 inches wide rather than the standard 14 inches by 17 inches. At times we have used a 34-inch film that folds, but even this does not take care of the need for a film long enough to take a picture of an entire tibia, a spine (scoliosis) or a femur. Has the time now come to redesign X-ray equipment to accommodate the taller patients we are seeing?

**Arthur A. Kirk, MD**

Orthopaedic Surgery, Ltd.  
3300 High Street, Portsmouth VA 23707

### **Roanoke Academy brochure fights drunk driving**

The articles on alcoholism in *VIRGINIA MEDICAL*'s November issue<sup>1</sup> prompt me to write about an educational program the Roanoke Academy of Medicine is sponsoring. It is aimed at drunk driving, which is such a menace on our highways, and it is designed primarily to augment the driver training programs of the 12 high schools in the Roanoke area.

Our primary tool is an illustrated brochure titled "A Message to My Patients," which is written as though a physician is speaking. There is information on crash factors; on social versus problem drinking; on such common myths as "Cold showers and black coffee help sober you up"; and other pertinent aspects of the problem. It is good-looking and professionally written.

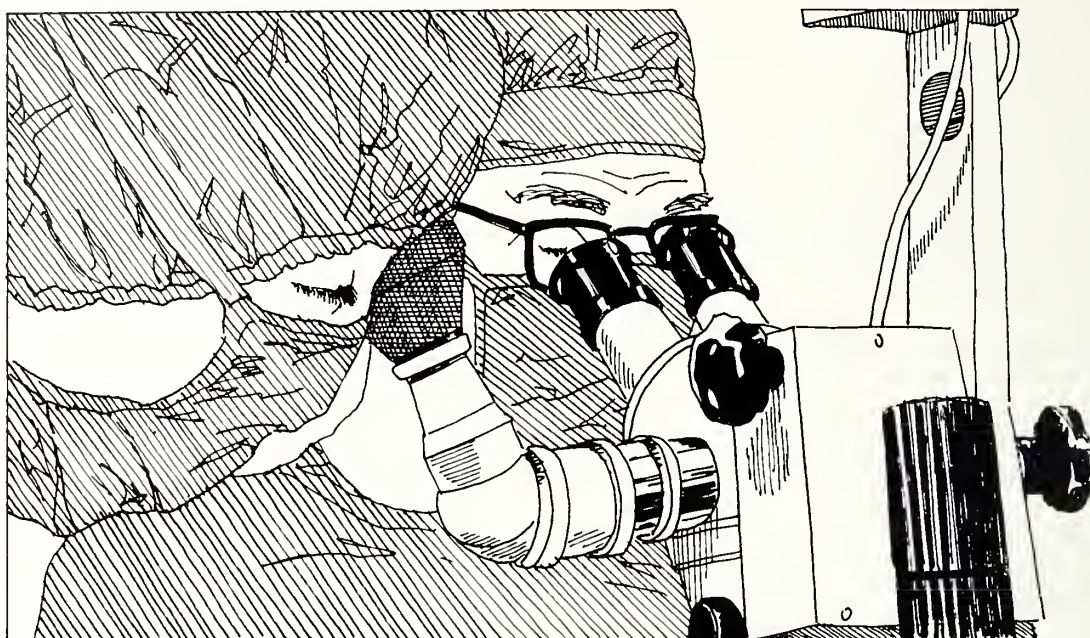
The plates for this booklet were provided to us free of charge by the US Department of Transportation, whose address appears on the end page, but the Roanoke Academy's logo and address is there, too. To print 4,000 of them cost us \$302.51. We distribute them free to the schools, and we have also offered them free to the public through radio and TV programs.

It occurs to me that other medical societies might like to undertake a similar project. I would be happy to send a sample of the brochure to anyone who is interested.

**Michael J. Moore, MD**

Lewis-Gale Clinic  
1802 Braeburn Drive, Salem VA 24153

1. Keiter RH: Helping alcoholics. Shires CL: From an alcoholic, a thank-you note. HVG: An impaired physician's story. *Va Med* 110:656-661, 1983



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# UP FRONT WITH THE PRESIDENT

## Communication and Participation

**I**N REFLECTING on 1984 one year from now, it is my hope that my tenure in office will be epitomized by two words—communication and participation.

These are qualities that are important every day to individuals of all walks of life and to all successful organizations. They are qualities that are particularly important to us as physicians and our organizations.

As physicians and as medical organizations, we have a tremendous need to communicate—among ourselves, with our patients and with the public and its leaders as a whole.

Communications is important, not only to avoid misunderstanding, undue concern and apprehension, but to foster a better comprehension of our philosophy, decision-making rationale and, perhaps most importantly, to instill confidence.

However, it is also important to remember that communications is a two-way street. It is incumbent upon our profession to demonstrate proficiency not only in communicating our views but also in listening to and appreciating the views of others.

As for participation, it is fair to say that in most organizations only 10%-15% of all members are actively involved. Another 40% can be classified as active supporters and an equal percentage are basically unconcerned, uninvolved and, in general, could care less. Unfortunately, there also is a final 10% or so that is basically negative, unconstructive and unsupportive.

In 1984 I would like to ask each Society member to assess his or her current status and resolve to move up, hopefully, into one of the first categories, and to lend knowledge, support, time and money to make The Medical Society of Virginia an even stronger, more effective, more responsive organization. I also urge you to take the same approach toward your county medical society and the Ameri-

can Medical Association.

Each of these organizations can benefit greatly from your active participation.

In early January I contacted all members of the Society's House of Delegates members and requested that they spend one or two days in Richmond during the current session of the General Assembly. It is important that state legislators recognize that medical leaders are concerned about the issues being discussed. It also is important that they recognize we are prepared, willing and interested in giving of our time and knowledge to improve the legislative process and arrive at better legislation, if needed, for the benefit of the citizens of our commonwealth.

It is my hope that all Society delegates will avail themselves of this opportunity to participate in and contribute to the legislative process and communicate their findings to the membership as a whole.

I have long been impressed by and applauded those delegations that have full delegate slates and frequently alternate delegates for both meetings of the House of Delegates at The Medical Society of Virginia's annual meetings.

Participation of this magnitude, coupled with communication with the membership as a whole, is essential to the health, strength and utility of our Society at all levels.

In short, opportunities to participate and have your voice heard in Society activities are plentiful. I urge you to take advantage of them.

If you are unable to be an active participant, be an active supporter. Be informed. Contribute positively. Work constructively to improve and strengthen your Society.

As the Uncle Sam recruiter sign says—Join Up! Get on the Team. We want (and need) YOU!

**C. Barrie Cook, MD**, President  
The Medical Society of Virginia

# OFFICERS OF THE LINE

*With a series of photographs of their presidents, VIRGINIA MEDICAL salutes the component medical societies, front line of organized medicine in Virginia. This is the third entry in the series.*



Presidents of the Southwestern Virginia Medical Society and the year each was elected: from left, Dr. Carl E. Stark, 1968; Dr. Joseph H. Early, Jr., 1967; Dr. Morgan E. Scott, 1975; Dr. George R. Smith, Jr., 1974; Dr. John Boniface, 1982; Dr. Donald B. Nolan, 1983; Dr. James Hal Smith, 1970.

**T**HE Southwestern Virginia Medical Society is one of the most venerable components of The Medical Society of Virginia. It was founded on July 22, 1852, according to an early, handwritten copy of the proceedings of the society's first meeting in Abingdon. This document from yesteryear was given to the author by Dr. Cecil C. Hatfield, Saltville, a former president of the Southwestern Society, and includes the original constitution and bylaws and a list of members.

There were 23 members at that first meeting, and they elected two Abingdon doctors to office: Dr. Daniel Trigg as president and Dr. James H. Dunn as secretary. Several honorary members are listed, including Dr. Robert W. Haxall of Richmond, who three times had been president of The Medical Society of Virginia, in 1841, 1842 and 1850; Dr. Hunter H. McGuire, then of Winchester, later of

Richmond, who became President not only of The Medical Society of Virginia but of the American Medical Association; and Dr. J. Marion Sims, who had just moved to New York from Montgomery, Alabama, and was on the threshold of his brilliant career in gynecology.

These honorary memberships presaged the Southwestern Society's custom of offering active, dues-paying membership to physicians outside of its geographical area. That hospitality accounts in part for the fact that 22 members of the Southwestern Society have served as Medical Society of Virginia presidents, eight of them from Roanoke, where they also belonged to the Roanoke Academy of Medicine.

Other factors contributing to this show of leadership are the Southwestern Society's early beginnings and its large size: Its geographical boundaries



are those of the 9th congressional district, which encompasses the ten counties of Bland, Carroll, Giles, Grayson, Montgomery, Pulaski, Russell, Smyth, Washington and Wythe.

The society existed in a number of forms and under a number of different titles over the years, accommodating its organizational structure to changing conditions. Local societies sprang up, such as the Abingdon Academy, after the Civil War, and the Wythe County Medical Society, which was in operation in 1877. Neither exist now.

Early Southwestern Society members were "ever active, and always the most numerous attendants upon the session of the state society," reported the December 1880 issue of the *VIRGINIA MEDICAL MONTHLY*.<sup>1</sup> Quarterly meetings were held at Marion, and free clinics, in which the poor came before the Society for examination, prescription, and operation if necessary, free of charge.<sup>2</sup>

Today the Southwestern Society schedules twice-yearly meetings. They are gatherings of friendly doctors who maintain personal and profes-

sional relationships while practicing over a vast area. In 1982 the society's roster listed 475 members.

Virginia's magnificent Southwest is pioneer country, the land of the Wilderness Road, the Cumberland Gap, and the Blue Ridge, Allegheny and Cumberland Mountains. In this beautiful setting, the Southwestern Virginia Medical Society has flourished for 130 years.

WILLIAM H. KAUFMAN, MD

1. Southwest Virginia Medical Society (editorial) *Va Med Mon* 8:751, 1880
2. Blanton WB: *Medicine in Virginia in the Nineteenth Century*. Richmond, Garrett & Massie, p 100, 1933

Address correspondence to Dr. Kaufman at 16535 Yeoho Road, Sparks, Maryland 21152, where he has been living in retirement since 1977. He previously practiced for 30 years in Roanoke, belonging to both the Southwestern Virginia Medical Society and the Roanoke Academy of Medicine.

## Fairfax Society celebrates big birthday

Twelve country doctors traveled by horse and buggy to the Fairfax County Courthouse on May 1, 1884, and organized themselves into the Fairfax County Medical Society.

Today the society's membership numbers 700 and there's not a country doctor in the bunch; Fairfax County is now thoroughly metropolitan, as is all of the "Upper Virginia" area contiguous to Washington. And when the society's members pull up to the high-rise Marriott Crystal City Hotel on October 13 for the centennial banquet, they will be driving horseless carriages.

The banquet is but one of many events in celebration of the 100th birthday. In April the society will sponsor a 10-kilometer "Run for Your Life." A reception at "Woodlawn" on the Mt. Vernon estate is scheduled for a day in May. As a birthday gift to the county, Fairfax Society physicians will conduct free blood pressure checks at stations set up in July and August at the county's four shopping malls. Nearing completion is a professionally researched and written

history of medicine in Fairfax County; a slide presentation developed from the material will be offered to the county's schools.

Records reveal that the Fairfax Society's early meetings followed the pattern of other component medical societies in Virginia—all-day sessions for the exchange of clinical information at members' homes, with the wives providing copious noon-time meals. In 1900, the proceedings relate, a paper on abortion was read; there followed lengthy and unresolved debate on the methods of performing abortions but no quarrel as to their legitimacy. When trolley transport arrived, the members would board a car at Vienna and ride to Washington to hear a highly regarded lecturer.

In this landmark year, Dr. C. Barrie Cook of Fairfax is President of The Medical Society of Virginia, the first Fairfax Society member to hold the post. Dr. Cook is a past president of the Fairfax Society. Dr. William L. Rich III is the current president.

A.G.

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# NEW MEMBERS



## *Accomack County Medical Society*

**Richard A. Szucs, MD**, General Practice, Box 2097, Oak Hall VA 23416

## *Albemarle County Medical Society*

**Joan F. Atkin, MD**, Genetics, Box 386, University of Virginia Medical Center, Charlottesville VA 22908

**Alfonso M. Lledo, MD**, Radiology, Box 170, University of Virginia Medical Center, Charlottesville VA 22908

## *Alexandria Medical Society*

**Stuart L. Davidson, MD**, Orthopedic Surgery, 1300 Lafayette Drive, Alexandria VA 22308

**Felix R. DiPinto, MD**, Internal Medicine, 6300 Stevenson Ave., Alexandria VA 22304

## *Augusta County Medical Society*

**Douglas W. McKibbin, MD**, General Surgery, 220 Rosser Ave., Waynesboro VA 22980

**William E. Rogers, MD**, Radiology, 362 S. Laurel Ave., Waynesboro VA 22980

## *Culpeper Medical Society*

**Robert B. Redmon, MD**, Otolaryngology, 610 Laurel St., Culpeper VA 22701

## *Fairfax Medical Society*

**Deborah G. Clapp, MD**, Pediatrics, 5640 North 19th St., Arlington VA 22205

**Paul T. Elder, MD**, Anesthesiology/Intensive Care, 6013 Conway Road, Bethesda MD 20817

**Joanne Herrman, MD**, Obstetrics/Gynecology, 8320 Old Courthouse Road, Vienna VA 22180

**Steven D. Larson, MD**, Family Practice, 2579 John Milton Drive, Herndon VA 22071

**John W. McFarland, MD**, Psychiatry, 3842 N. Dittmar Road, Arlington VA 22207

**Murray B. Welt, MD**, Emergency Medicine, 1111 Arlington Blvd., Arlington VA 22209

## *Fauquier County Medical Society*

**Norman L. Mauroner, Jr., MD**, Internal Medicine, 328 Hospital Hill, Warrenton VA 22186

## *Lynchburg Academy of Medicine*

**Pamela W. Wallace, MD**, Psychiatry, 2250 Murrell Rd., Lynchburg VA 24501

## *Newport News Medical Society*

**W. L. Old III, MD**, Vascular Surgery, 11030 Warwick Blvd., Newport News VA 23601

## *Norfolk Academy of Medicine*

**Paul Walker, MD**, Pediatrics, 2501 Marshall Ave., Newport News VA 23607

## *Northampton County Medical Society*

**Robert M. Paschall, MD**, Neurology, NAM Hospital, Nassawadox VA 23413

## *Northern Neck Medical Society*

**James F. Hamilton, MD**, Obstetrics/Gynecology, Box 880, Kilmarnock VA 22482

## *Portsmouth Academy of Medicine*

**Reymond G. Pascual, MD**, General Surgery, 622 Citizens Trust Bldg., Portsmouth VA 23704

**Federico Vizcaino, MD**, Psychiatry, 355 Crawford Pkwy, Portsmouth VA 23704

## *Richmond Academy of Medicine*

**Daniel A. Dethmers, MD**, Orthopedics, 2911 Grove Ave., Richmond VA 23226

**Alexander K. Girevendulis, MD**, Diagnostic Radiology, 606 Lambeth Road, Richmond VA 23225

**John J. Hennessey IV, MD**, Neurology, 3249 Summerbrooke Dr., Richmond VA 23235

**John W. Hyslop, MD**, General Surgery, 3536 Grove Ave., Richmond VA 23221

**Donald A. Taylor, MD**, Pediatric Neurology, 1825 Monument Ave., Richmond VA 23220

## *Roanoke Academy of Medicine*

**Mark R. Hanabury, Jr., MD**, Otolaryngology, PO Box 1789, Roanoke VA 24008

**Gershon Silber, MD**, Psychiatry, PO Box 3036, Roanoke VA 24015

## *Tri-County Medical Society*

**Thomas D. Salyer, MD**, Obstetrics/Gynecology, 707 Gittings St., Suffolk VA 23434

## *Virginia Beach Medical Society*

**Joan W. Devine, MD**, Obstetrics/Gynecology, 1288 Paramore Dr., Virginia Beach VA 23454

**Ramon N. Redford, Jr., MD**, Gynecology, 1120 First Colonial Rd., Virginia Beach VA 23454

**Douglas R. Schreiber, MD**, Orthopedic Surgery, 1100 First Colonial Rd., Virginia Beach VA 23454

## *Williamsburg-James City Medical Society*

**Leland Gerde, MD**, Ophthalmology, 111 Albemarle Ave., Williamsburg VA 23185

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# Head Injuries, Part I: Solving Pre-Hospital Care Problems

Joseph B. Warren, BSN, John D. Ward, MD, and Donald P. Becker, MD,  
*Richmond, Virginia*

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Secondary insults that occur before the head-injured patient arrives at the emergency department may significantly affect outcome. The authors analyze the pre-hospital care of 100 head-injured patients and formulate specific therapies that can improve the survival rate.

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**H**HEAD INJURIES contribute significantly to the mortality and morbidity associated with trauma. This is especially true in patients in motor vehicle mishaps, where at least 50,000 of the accidental deaths are due to the automobile and 60-70% have evidence of severe brain injury.<sup>1</sup>

There are a number of factors which have previously been implicated as worsening the outcome from severe head injury. Most of these secondary systemic insults occur before the patient's arrival at the emergency department and may well be major contributions to outcome in these patients regardless of subsequent treatment.<sup>2</sup>

In order to characterize the effectiveness of pre-hospital care afforded to these patients and to

document the occurrence and extent of secondary insults with head-injured patients, the present study was undertaken. The importance of prompt transfer to an appropriate trauma center, based on the paramedic's evaluation of the extent of brain injury, was also evaluated, as was a description of the clinical status of the patients upon arrival in the hospital. We have analyzed more closely the significance of pre-hospital assessment and management and noted specific emergency medical therapies which will improve outcome from head injury.

## SECONDARY SYSTEMIC INSULTS

There is a wide continuum of head injury, with a wide diversity of neurologic abnormalities. These may vary from a simple scalp laceration to an extensive diffuse brain injury. Skull injuries, per se, are not of great significance except in relation to any brain damage they cause, intracranial bleeding they create, or infection they may lead to. The injury to the brain represents the most serious sequel to head trauma.<sup>3</sup>

From the Division of Neurosurgery, Department of Surgery, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Becker at Box 631, MCV Station, Richmond VA 23298.

This is the first in a two-part series. Part II is to appear in VIRGINIA MEDICAL's April 1984 issue.

Submitted 11-24-83.



Beyond the primary head injury, that occurring at the time of impact, is a series of events which contribute to further injury. These include systemic hypotension, alterations in blood gases (hypoxemia, on hospital admission  $\text{PaO}_2 < 65$  mmHg and/or hypercarbia, a hospital admission  $\text{PaCO}_2 > 45$  mmHg), intracranial hypertension and infection, which represent a "second" head injury.<sup>4</sup> Further brain damage occurs after there is delayed bleeding inside the skull, resulting in an intracerebral hematoma or epidural or subdural hematoma. The further damage to the brain from delayed bleeding is due to shifts of brain and increasing intracranial pressure. These events compromise cerebral blood flow (CBF) and cerebral oxygenation at a time when the mechanisms for adaptation to these additional stresses may be impaired by the effects of the head injury on the cerebral vasculature.<sup>5</sup>

The effects of secondary systemic insults on outcome have been described in several series. Miller et al in 1978 indicated that secondary systemic insults (hypoxia, hypotension, anemia, hypercarbia) after severe head injury were all associated with a worse outcome than similar injuries without systemic insults. These insults were almost exclusively associated with multiple injuries and were caused usually by vehicular accidents.<sup>2</sup> Rose, in a review of 116 patients who verbalized after head injury and subsequently died, demonstrated one or more avoidable factors which contributed to the death in 54% of these patients, with a delay in treatment of an intracranial hematoma the most common factor.<sup>6</sup> Hypoxia and systemic hypotension were also mentioned prominently.

Jennett and others in a 1981 study of 150 patients transferred to a neurotrauma center showed that untoward incidents which caused secondary brain damage were present in 61 patients (41%) and that extracranial injuries were overlooked or inadequately treated in 21 patients.<sup>7</sup> The most common areas mistreated were airway destruction and hypotension. In a study by Seelig et al, delay from injury to operation in 82 patients with traumatic acute subdural hematomas was the factor of greatest therapeutic importance.<sup>8</sup> Patients who underwent surgery within the first four hours after injury had a 30% mortality rate, as compared to 90% in those who had surgery after four hours ( $p < 0.0001$ ). Commenting on the Seelig article, Freeark, in the 1982 American Association for the Surgery of Trauma presidential address, stated that a period of two hours makes the difference between a functioning brain and a hopeless invalid and that any emergency medical service (EMS) that fails to place head

injuries in neurosurgical treatment centers in a matter of minutes is not doing its job.<sup>9</sup>

## MATERIALS AND METHODS

We prospectively examined the records of 100 consecutive patients with severe head injury who were admitted to the neurosurgery service at the Medical College of Virginia (MCV) from January 1980 through December 1981. The mean age was 31 years old, and 78 males were in this series. After appropriate non-surgical resuscitation, all patients included in this study were unable to speak intelligible words, even after noxious stimuli, and were unresponsive to verbal commands. General and neurologic evaluations were undertaken, and blood for arterial blood gases, electrolyte and hematology studies, drug and toxic-substance screens, and ethanol were drawn. Endotracheal intubation was accomplished in all patients after neurological examinations, unless the patient was in respiratory distress.<sup>3,10</sup>

Detailed information concerning the pre-hospital phase of treatment for each patient was obtained by direct observation, by questioning rescue squad personnel, and by scrutinizing the transfer records which accompanied the patient. Where data were confused or doubtful, the referring hospital or rescue squad was contacted for more details within 24 hours of admission to MCV.

## RESULTS

Following the method of Champion et al and others,<sup>11-13</sup> assessments of the severity of injury and the effects of pre-hospital therapy were made by measuring the Glasgow Coma Scale (GCS) and Trauma Score (TS) for each patient. Retrospectively, both the GCS and TS for each patient were calculated and then compared to the GCS and TS measured upon arrival to the emergency department, and changes in clinical status noted. Where deterioration of either score was seen, factors responsible for the decline were indicated.

The Trauma Score is one commonly used index of clinical injury severity<sup>14</sup> and is also used to project probability of survivability based on levels of TS.<sup>15</sup> The TS at the first paramedic evaluation was compared to the TS at the MCV emergency department, and the differences tabulated. There was no change in slightly more than one-half of the patients, while 20 showed a decrease in TS and 24 improved in TS. Of the 20 patients who declined, nine had operable hematomas, six suffered signifi-

**Table 1. Systemic Insults Observed on Admission in 100 Head-Injury Patients.**

Systemic Insult	Percent	MVA (%)	Poor Outcome (%)
Arterial hypoxemia (PO <sub>2</sub> <60 mmHg)	29	78	54
Arterial hypotension (SBP <90 mmHg)	16	94	48
Anemia (hematocrit <30%)	8	65	65
Arterial hypercarbia (PCO <sub>2</sub> >45 mmHg)	10	78	78
Multiple injuries	45	79	59
None	48	70	38

MVA = associated with motor vehicle accident

SBP = systemic blood pressure

Poor outcome includes severe disability, vegetative state and death.

Multiple injuries = two or more additional injuries (abdominal, chest or long bone)

cant multiple trauma (abdominal or chest injury requiring surgery), three were hypotensive (systolic blood pressure less than 90 mmHg at MCV) secondary to hypovolemia, and two patients had respiratory embarrassment related to mechanical obstruction of the airway.

Based on the estimate that the Trauma Score does not reflect well the contributions of CNS trauma to the overall TS, we plotted the Glasgow Coma Score at the same intervals as the TS. (Our estimate is based on the 87% estimated survival possible in a patient with a TS of 12 and a GCS of 3—flaccid, not opening eyes, and not vocalizing—and all other parameters of the TS normal). From initial paramedic evaluation to presentation in the MCV emergency department, 33 patients improved during the pre-hospital phase of management and transportation, 40 patients had no change in GCS, and 27 patients declined. Of the 27 patients who declined, 11 had multiple injuries, chiefly abdominal and chest which required surgical intervention, nine had operable intracranial mass lesions (five acute subdural hematomas, three epidural hematomas, and one intracerebral hematoma), four had respiratory difficulty upon admission, and three were hypotensive secondary to hypovolemia. All four patients with respiratory difficulty also had admission PaO<sub>2</sub> levels less than 60 mmHg, and none had airways during transportation.

The differences between TS and GCS in describing the clinical status of these head-injured patients were compared with respect to changes in score, as an estimate of the effects of pre-hospital care. The difference between the TS and GCS was significant when analyzed by the chi-square test ( $p < 0.001$ ).

Hypoxemia on arrival was noted in 29 patients

and was associated with airway obstruction by aspirated blood, vomitus, or secretions in 22 (76%) patients (Table 1). During transfer from referral community hospitals or from the scene of the accident, 21 patients had an endotracheal tube in place, 52 had oropharyngeal airway, and 27 patients had no airway. All patients with endotracheal tubes in place were transferred from other hospitals, and six (29%) were hypoxic. Similarly, hypoxemia was noted in eight patients with oropharyngeal airways (15%), and 15 patients with no airways (56%). For the whole series, patients who were hypoxic had a worse outcome than those who had normal PaO<sub>2</sub> on admission ( $p < 0.001$ ).

Sixteen patients were hypotensive (systolic blood pressure <95 mmHg) on admission. All had suffered significant systemic injury (long bone fracture, major chest injury, abdominal visceral injury) in addition to their head injury, and all but two had suffered vehicular accidents. Intravenous fluids had been administered in the field to five patients, all of whom had been transferred from local receiving hospitals. An additional 18 patients were noted to have admission SABP readings of 96 to 105 mmHg; half of them had been in motor vehicle accidents.

When defined by type of head injury, 40 patients had significant intracranial mass lesions requiring surgical intervention, while the remainder had diffuse injuries. Important elevations in the intracranial pressure above 20 mmHg were noted in about two-thirds of the patients. Intracranial hypertension was seen in 80% of the patients with mass lesions, as compared to only 57% in patients with diffuse injury (Table 2).

## Outcome

The outcome of all patients was assessed at three months, six months or one year after injury, using the Glasgow Outcome Scale (GOS).<sup>17</sup> Overall, 57% of the patients made a good recovery, 20% were moderately disabled but able to care for themselves, 4% remained severely disabled requiring the help of others and 39% died. Patients in the surgical group had a higher mortality (58%) than patients in the non-surgical group (26%) ( $p < 0.001$ ). The

**Table 2. Intracranial Pressure According to Type of Injury in 100 Head-Injured Patients.**

	ICP-normal	ICP-raised but reducible	ICP-raised, not reducible	
Mass lesion	7 (8%)	16 (18%)	12 (13%)	35
Diffuse injury	24 (26%)	26 (28%)	6 (7%)	56
Total	31 (34%)	42 (46%)	18 (20%)	91

% = % of total.



highest mortality was noted in patients with acute subdural hematomas (63%). Within the non-surgical group, worse outcome (severe disability, vegetative survival and mortality) was more frequent in patients with cerebral contusion (48%) than in patients with diffuse brain injury (31%) ( $p < 0.01$ ).

## DISCUSSION

There is little doubt that patients with severe head injury present a management problem for pre-hospital personnel, especially in areas where there is no regional transportation protocol. A study by West and Trunkey comparing a regional emergency medical system (REMS) and a non-organized system revealed that 28% of the CNS-related fatalities were preventable in the non-organized system.<sup>16</sup> The practice in many areas of simply transporting patients with head injuries to the nearest medical facility, regardless of that facility's ability to adequately treat this population, may jeopardize maximum outcome in many instances.

The preponderance of secondary systemic insults in this group of patients is consistent with findings from our earlier studies, and that of other investigators<sup>2,6,7,10,13,14,16,17</sup> (Table 1). This high incidence of systemic injury to an already damaged brain is commonplace in that critical early period between injury and admission to the hospital, a period of "therapeutic vacuum," and needs to be addressed clearly in pre-hospital management protocols. We find that arterial hypotension and anemia are closely associated with multiply injured patients. Hypotension is seldom caused by head injury alone but generally is related to hypovolemia, chest injuries, associated spinal injuries or septic shock.<sup>17</sup>

Hypoxia closely follows a depression of consciousness in most every case, which leads to an inability to maintain an adequate airway. Mechanical obstruction was noted by the rescue squads in about one-third of the patients, most often due to blockage of the mouth and pharynx by secretions and blood.

Brain injury frequently interferes with brain-stem respiratory centers by pressure from above the tentorium or localized brain injury, and the patient "forgets to breathe." This leads to inadequate ventilation and concurrent suppression of cough and gag reflexes.<sup>5</sup> Placement of any airway and administration of oxygen in transport lessened the incidence of hypoxia in our series, indicating that this simple step is useful and important. It is our policy to intubate any patient who does not obey commands after a head injury, on the assumption

that a brain injury which renders the patient unable to obey verbal commands is severe enough to cause brain-stem abnormalities.

The incidence of shock in these patients is similar to reports elsewhere and may reflect emergency medical service capabilities during the study period, when pre-hospital personnel were not permitted to begin intravenous fluids. Hypovolemia can be suspected in that 80% of the hypotensive patients were also anemic (hematocrit  $< 30\%$ ). The causes of shock for this group were chest and abdominal injuries and hypovolemia, suggesting that aggressive fluid resuscitation and rapid transfer would have influenced hypotension. The reluctance to give adequate fluids may have been based either on an underestimate of fluid needs or a fear of precipitating cerebral edema. While the latter is possible, cerebral edema generally develops more than six hours after injury.<sup>17</sup>

The differences between Trauma Score and Glasgow Coma Scale is important in these patients. In this regard, the TS suffers from the same difficulty in describing the implications and consequences of severely head-injured patients as other anatomical indices of injury severity, which all underestimate the effects of the CNS injury. The use of the TS or

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See also the editorial on page 106.

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other such scales which under-predicts mortality and morbidity after head injury as pre-hospital determinants of treatment facility priorities may mean that the treatment facility is under-prepared to meet the needs of these patients.<sup>11</sup> The hypothetical patient with a TS of 12 and GCS of 3 has an 87% probability of survival according to the former and an 87% incidence of death from the latter.<sup>15,19</sup> Clearly, this indicates that placing these patients in an inappropriate facility may endanger their lives.

The importance of immediate transfer of patients with significant head injuries to the proper neurosurgical facility can also be inferred from these studies and others. It appears from our data that a temporary stop at another hospital en route to the Level I trauma center does not benefit patients, since the incidence of secondary systemic insults in patients referred from another hospital was just as high as the percentage in patients who came directly from the scene of the accident. A lower incidence of systemic insults was reported from a neurotrauma center in California where all injuries were routed to a small number of designated neurosurgical centers and where the paramedical service was particularly efficient.<sup>18</sup> We believe that the only indicators for stopping at a community receiving hospital en route

to the neurotrauma center are severe systemic hypotension unresponsive to therapy or an airway that is endangered.

Raised intracranial pressure was an important adverse factor, especially for death. Severe intracranial hypertension (more than 40 mmHg) in patients with head injury appears directly responsible for brain ischemia and severe, if not fatal, neurological dysfunction.<sup>10</sup> In our series, 65% of the 91 patients monitored had a maximum ICP greater than 20 mmHg, and all had a worse outcome than those who had a "normal" ICP of less than 20 mmHg, when measured by the Glasgow Outcome Scale. Since ICP monitoring is commonplace almost exclusively at neurotrauma centers, it would seem important to transport head-injured patients to facilities where this portion of management, by experienced physicians and nurses, is available.

While the time of transportation of head-injured patients has been shown to be important in decreasing mortality and morbidity, we do not suggest that prompt transportation of this population be done at the expense of proper extrication and emergency care. Fully one-third of our patients traveled over 50 miles en route to the Medical College of Virginia, and rescue squad estimates, although rough, of duration at the scene were from 15-30 minutes to extricate the patient, administer oxygen, stabilize the spine, start intravenous line, splint fractures, dress wounds and so forth. This agrees well with the estimates of Hedges, Sacco et al for patients with blunt trauma.<sup>12</sup> The mean time duration between injury and the time the patient was seen by an attending neurosurgeon at MCV was 2.77 hours, with 78% arriving in less than 4 hours. However, delays in transportation or at a receiving hospital can be devastating, especially in patients with intracranial mass lesions who have higher ICP courses, greater instances of abnormal brain-stem function and increasing age, all factors important in predicting a poor outcome.<sup>8,10</sup>

### CONCLUSION

Optimal pre-hospital care of comatose patients requires rapid transfer to appropriate neurosurgical trauma units with proper emergency facilities, computerized tomography, immediately available operating rooms, in-house neurosurgeons, and a neuroscience ICU with trained, experienced nurses. There is little doubt that if patient deterioration is avoided in the pre-hospital phase, through assessment, management and transport, the patient survival rate will be maximized.

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# Vascular Leiomyoma of the Superior Mesenteric Vein

John T. Mazzeo, MD,  
Woodbridge, Virginia, and  
Marion Jordan, MD, Washington, DC

**T**UMORS of the smooth muscle arising from a large vein are exceedingly rare. As of 1960, Thomas and Fine<sup>1</sup> had reported only 29 cases of primary large venous tumors; 21 were of smooth muscle origin and of these, only eight were benign. In the intervening period, there has been reference to only two other benign smooth muscle venous tumors, one arising from the renal vein<sup>2</sup> and the other from the internal saphenous vein.<sup>3</sup> To the author's knowledge, this is the first reported case of a vascular leiomyoma from the superior mesenteric vein.

## Case Report

K.B., a 66-year-old white female, was admitted to the hospital April 23, 1976, with a two-month history of vague abdominal discomfort. Past history was essentially non-contributory. Physical examination revealed a non-tender abdominal mass, 10 × 8 cm in diameter. The mass was easily ballotable and did not appear fixed either to intra-abdominal contents or the abdominal wall. Diagnostic evaluation, including sigmoidoscopy, barium enema, upper gastrointestinal series and intravenous pyelogram, was normal. Abdominal ultrasound demonstrated a 6-cm solid mass adjacent to the

Address correspondence to Dr. Mazzeo at 1982 Opitz Boulevard, Woodbridge VA 22191.

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Fig. 1. Arteriogram shows tumor mass with venous drainage into superior mesenteric vein.

superior mesenteric vessels. A superior mesenteric arteriogram was performed with particular attention to the venous phase. Multiple tumor vessels were seen within a confined tumor mass, with venous drainage directly into the superior mesenteric vein (Fig. 1).

On April 30, 1976, exploratory abdominal laparotomy was performed. A large mass was found within the mesenteric leaves of the proximal jejunum, adjacent to the superior mesenteric vein. Five centimeters of distal superior mesenteric vein, a portion of the jejunal mesentery, and two feet of proximal jejunum (devascularized during tumor extirpation) were removed in continuity with the abdominal mass. The tumor was attached directly to the portion of the superior mesenteric vein distal to the major jejunal vessels and next to the major ileal vessels; thus resection could be done without compromise to the remaining intra-abdominal contents or surgical involvement of the remaining superior mesenteric vein. The patient had an uneventful postoperative course with no evidence of recurrence in the subsequent six years.

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### **Pathology**

The gross specimen was  $9.5 \times 7.0 \times 4.5$  cm. The mass was solid and multi-lobulated and, in transection, showed a pale tan soft tissue. In addition, there was a 5-cm segment of vein,  $1.1 \times 0.8$  cm in diameter, extending from the periphery of one of the lobulated areas. The mass appeared encapsulated by a thin, gray fibrofatty vascular membrane, which was intact and adherent to the wall of the vein; it was stripped from the underlying tumor mass with only moderate difficulty. A 40-cm segment of jejunum and accompanying mesentery were submitted with the specimen.

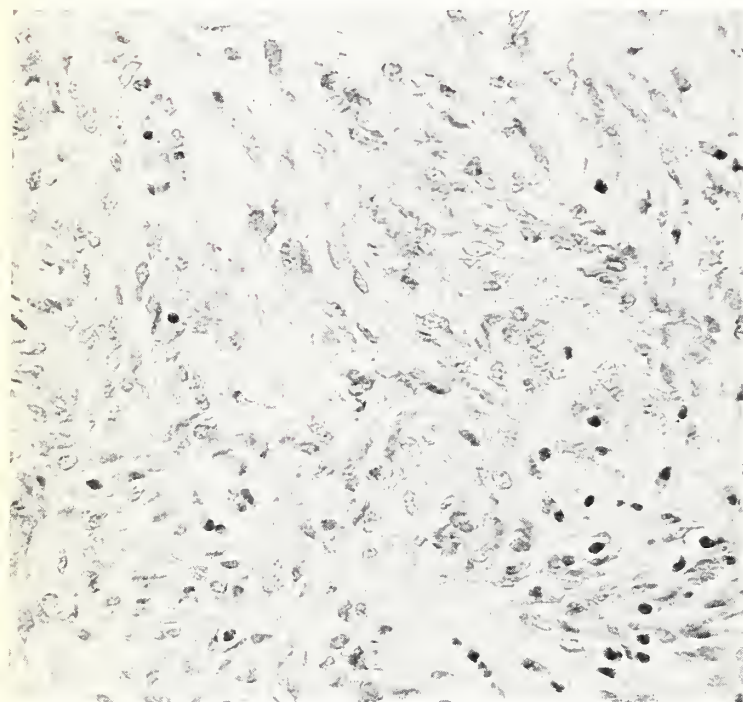
Microscopically, the tumor was a solid neoplasm constructed of whorled fascicles of spindle cells arranged in a definite angiocentric distribution. Individual cells exhibited fusiform non-pleomorphic nuclei with minimal mitotic reduplicative activity: 2-3 mitoses per highpowered field (Fig. 2). Multiple sections revealed the tumor to be completely encapsulated with no evidence of invasion into the external mesenteric adipose tissue. The tumor was therefore reported as a vascular leiomyoma of the superior mesenteric vein.

### **Discussion**

Vascular leiomyomata are by definition benign tumors arising from the smooth muscle of blood vessels. Because of the small number of reported cases, more definite criteria have been difficult to formulate. DeWeese<sup>4</sup> and Haug and Losli<sup>5</sup> in 1954 could only demonstrate two case reports where the tumor was actually seen to originate from the vein. Ackerman and Del Regato<sup>6</sup> even questioned diagnoses of benignity, emphasizing the ability of these tumors to occasionally act in a malignant and invasive fashion even when previously reported as benign.

Therefore, in reporting a vascular leiomyoma of the superior mesenteric vein, we have evaluated the different criteria and rationales that have been used to classify all such vascular leiomyomas and then correlated them to this tumor. In addition, we are presenting the use of venous phase arteriography for pre-surgical treatment evaluation.

Of the ten benign tumors previously described, it was not always possible to observe the tumor originating from the smooth muscle of the vein. We also were not able to demonstrate this continuity microscopically, even though intra-operatively there was gross attachment of the tumor to the wall



**Fig. 2. Photomicrograph shows vascular leiomyoma with whorled fascicles of spindle cells. ( $\times 100$ )**



of the vein. There are some observers who have hypothesized that this is due to tumor growth occurring first in the perivascular connective tissue, as seen in small cutaneous vascular leiomyomas.<sup>7</sup> Then, in response to the proper stimulus, such as trauma or infection, this tissue has the embryonic ability to form smooth muscle, and coalesce with the muscle of the vessel wall. This concept may explain the problems that many authors have had in discovering direct venous smooth muscle involvement.

Pathologic classification may be difficult in separating the true vascular leiomyoma from other benign tumors, such as venous hemangiomas and hemangiopericytomas. The differentiation depends on the amount of smooth muscle present.<sup>9</sup> Hemangiomas may occasionally be the starting point of such tumors, with minimal non-striated muscle proliferating until a solid leiomyoma is achieved with minimal vascularization and a predominance of smooth muscle.<sup>10,11</sup>

Although this may sometimes be an arbitrary categorization, there was complete agreement as to the diagnosis of the tumor we excised.

The cytologic type has occasionally been seen to change with apparent well-differentiated and localized tumors developing progressive malignant characteristics such as invasion and metastases. It has been suggested that some benign vascular leiomyomas may not be an end-point but rather a mid-point in a process which ends in malignancy.<sup>7</sup> In an attempt to avoid this confusion, Varela-Duran, Oliva and Rosai<sup>12</sup> correlated microscopic features and biologic behavior. In those tumors which had less than 20 mitoses per highpowered field, there was no evidence of either local or distant recurrence. There was every indication that higher mitotic counts signify greater possibilities of developing metastatic disease; however, reduced mitoses did not always mean benignity, as the contrary had occasionally been proven.<sup>13</sup> So, even though our patient had only three mitoses per highpowered field, this classification could only be used as a general guide.

Therefore, treatment should always be early and consist of as wide a surgical excision of the tumor mass, adjacent vessels and peri-connective tissue as possible. Because of the concern over adequate collateralization and extension of tumor mass, preoperative angiography can be of great assistance in

the surgical approach.<sup>4,14</sup> In the relatively common sites of the inferior vena cava or peripheral venous system, phlebography should be the study of choice. However, with an abdominal lesion of uncertain origin, venous phase arteriography, in our case via the superior mesenteric artery, would be suggested. We were able to adequately assess the site of origin, tumor extent and collateral arterial and venous flow. This allowed safe surgical extirpation and uneventful recovery.

## Summary

A case of vascular leiomyoma of the superior mesenteric vein is reported. It is believed to be the only report of such a tumor primary in this vein. The pathologic and angiographic criteria for the diagnosis and treatment are reviewed.

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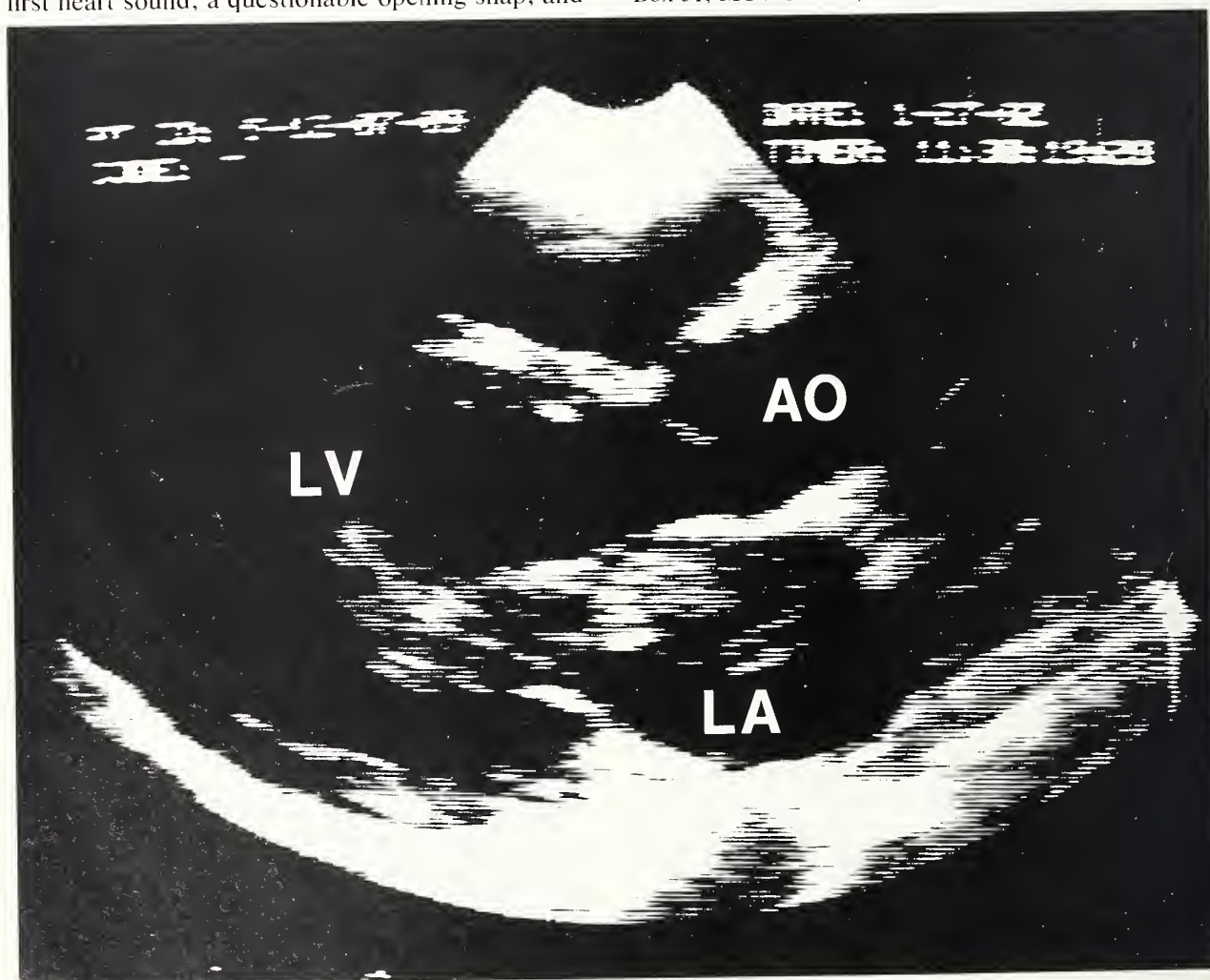
# Can You Diagnose This?

A medical puzzle prepared by  
Walter Paulsen, MD, *Richmond, Virginia*

This cross-sectional, or two-dimensional, echocardiogram was performed on a 34-year-old woman who presented with a five-month history of progressively declining exercise tolerance, paroxysmal nocturnal dyspnea, and intermittent lightheadedness. Cardiovascular examination disclosed a loud first heart sound, a questionable opening snap, and

a grade 2/6 mid-to-late diastolic murmur at the apex. An electrocardiogram was normal and a chest roentgenogram revealed left atrial enlargement.

From the Department of Medicine, Division of Cardiology, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Paulsen at Box 51, MCV Station, Richmond VA 23298.



DIAGNOSIS/DISCUSSION ON PAGE 97



# ABSTRACTS

These are abstracts of papers to be presented at the 1984 regional meeting of the Virginia Chapter, American College of Physicians, in conjunction with the Virginia Society of Internal Medicine, on February 24-26 at the Homestead in Hot Springs. Dr. Charles L. Crockett, Jr., is program chairman.

**Serological Markers for Hepatitis B in Hospital Personnel in a Moderate-Sized Community.** J. Boyd Francis, MD, *Roanoke*, Charles Schleupner, MD, and Douglas Blevins, MD, *Salem*.

Previous reports have described the prevalence of Hep B serologic markers to be 15-20% in health care workers, with rates as high as 30% or more in certain high risk subgroups. These reports frequently arise from large urban medical centers. To investigate the seroprevalence of Hep B markers among health care workers in a moderate-sized community (250,000), we conducted a serosurvey of personnel at the 4 acute-care hospitals in the community. Using antibody to Hep B surface antigen (anti-HB<sub>s</sub>) or both, we prospectively surveyed a total of 364 hospital employees working in areas of high risk. One hospital retrospectively examined the seropositivity of 90 employees who sought post-exposure care for accidental high-risk blood exposures (needle sticks, etc.). The results:

	No. of Employees	Serologic Marker	Sero- positive No. (%)
Hospital 1	138	anti-HB <sub>s</sub>	9 (6.6)
Hospital 2	84	anti-HB <sub>c</sub>	7 (8.3)
Hospital 3	90	anti-HB <sub>c</sub>	3 (3.3)
Hospital 4	52	anti-HB <sub>s</sub>	1 (1.9)
Retrospective	90	anti-HB <sub>s</sub>	3 (3.3)
TOTAL	454		23 (5.1)

Since the majority of previously sensitized patients should demonstrate both anti-HB<sub>s</sub> and anti-HB<sub>c</sub>, the majority of seropositive personnel should have been identified in this study. We conclude that the Hep B seroprevalence is lower in this medical community than that commonly reported in the literature. Guidelines for the administration of Hep B vaccine may require local modifications.

**Anticonvulsant-Induced Hepatocellular Necrosis and Severe Exfoliative Dermatitis.** John D. Malone, MD, *Virginia Beach*, and Rocco J. Volpe, MD, and Joseph E. Hancock, MD, *Portsmouth*.

Hepatocellular necrosis and severe exfoliative dermatitis are rare life-threatening complications of anticonvulsant therapy.

A 24-year-old black female developed exfoliative

dermatitis and hepatocellular necrosis (total bili 27, SGOT-944, prothrombin time 23 corrected to 15 with Vit K, following 6 weeks of diphenylhydantoin therapy. Two months into her convalescence on seizure medication she developed a tonic clonic seizure and phenobarbital therapy was initiated. Three days later pruritis, subcutaneous edema and eosinophilia developed along with an elevation of SGOT from 66 to 94. Phenobarbital was discontinued and carbamazepine therapy immediately initiated. After 5 days, carbamazepine was discontinued due to increasing subcutaneous edema, pruritis and recurrent exfoliative dermatitis. Valproic acid was prescribed and the patient's condition improved. In anticonvulsant therapy the potential for increased sensitivity to chemically related and unrelated pharmaceutical agents exist. Once a drug sensitivity is demonstrated, patients must be monitored closely with introduction of each new medication.

**Prevalence and Clinical Significance of Residual Myocardial Ischemia Two Weeks after Acute Non Q-Wave Infarction.** Robert S. Gibson, MD, Cheryl M. Curling, BSN, Richard S. Crampton, MD, and George A. Beller, MD, *Charlottesville*.

Despite better LV function, patients with non Q-wave infarction (NQMI) appear to have a comparable or worse prognosis than those recovering from Q-wave MI (QMI). This may be due to residual myocardium at risk in the perfusion zone of the infarct vessel. A higher incidence of thallium 201 (TI) redistribution and reinfarction or death after NQMI vs QMI would support this. Accordingly, we prospectively studied 160 consecutive patients with MB-CK confirmed acute MI, 47 with NQMI and 113 with QMI. All underwent predischarge exercise testing, quantitative TI scintigraphy and coronary angiography (angio) 11 ± 3d after MI. The Norris Index and prevalence of multivessel CAD by angio was similar in the 2 groups despite less necrosis with NQMI than QMI, reflected by lower peak CK (420 ± 277 vs 1122 ± 789,  $p < .001$ ) and higher LVEF (53 ± 10 vs 47 ± 11%,  $p < .003$ ). Compared to QMI patients, those with NQMI had more angina during exercise testing (12% vs 26%,  $p < .05$ ), more

nonoccluded infarct vessels (29% vs 55%,  $p < .01$ ) more T1 redistribution in the infarct zone (23% vs 55%,  $p < .001$ ) and a higher incidence of reinfarction or death (7% vs 21%,  $p < .01$ ) during  $20 \pm 11$  month follow-up. Thus, despite a smaller infarct size reflected by CK values and a better LV function, patients with NQMI have more residual myocardium at jeopardy than QMI.

**Acute Intervention in Myocardial Infarction, a Rural Hospital Experience.** John H. Cook III, MD, *Leesburg*, and Kenneth M. Kent, MD, *Washington, DC*.

Acute intervention in myocardial infarction has come into common use over the last two years. The high technology involved has restricted this service to the tertiary hospital setting. A joint protocol developed by the authors has provided timely access to acute intervention to patients in the rural setting. Intervention at Georgetown University Hospital has included streptokinase infusion, coronary angioplasty and emergency cardiac surgery. Analysis of the 17 cases to date has not revealed any significant departure from expected morbidity or mortality. There has been one death. Three patients have required defibrillation prior to arrival at Georgetown. Several valuable lessons in the areas of emergency medical services, local emergency room performance, intensive care unit organization and catheterization laboratory logistics have been learned. While the long-term utility of acute intervention in myocardial infarction remains to be proven, this early experience suggests that the radius of rapid access to high technology services can be safely expanded with minimal expense and moderate local reorganization.

**Therapeutic Parathyroid Autotransplantation in Renal Osteodystrophy (RO).** Newell R. Falkinburg, MD, Bruce D. Baird, MD, Robert E. Berry, MD, and Susan N. VandenBerg, MD, *Roanoke*.

Significant numbers of patients on hemodialysis today are experiencing the sequelae and complications of long-term dialysis, namely RO from secondary hyperparathyroidism (HP). This is usually a result of dietary as well as medical noncompliance. Our approach in treatment of RO and pruritis secondary to renal failure is total parathyroidectomy with transplantation (TPA). To date we have performed 16 radical parathyroidectomies with autotransplantation of a portion of the most normal-appearing gland into the brachioradialis muscle; 12 of the transplants have been for secondary HP, 4 for primary HP with diffuse hyperplasia. This report deals only with the secondary HP patients. These

patients have shown dramatic clinical improvement with loss of bone pain and pruritis as well as radiographic improvement of hand X-rays. Pathological examination of all parathyroid glands removed has revealed hyperplasia. Graft function has been documented by comparing serum parathyroid hormone levels of the grafted and nongrafted forearm. At present, our longest followup is 31 months and we have experienced minimal morbidity and no mortality related to the procedure. We have also found gland localization with preop CT scan to be of no value. Real-time ultrasonography has been of benefit in parathyroid gland localization. We conclude that TPA can be performed with minimal morbidity and limited hospital stay in the patient with RO.

**Massive Pleural Effusion Secondary to Continuous Ambulatory Peritoneal Dialysis (CAPD).** Antonia M. Harford, MD, Barry B. Kirschbaum, MD, Domenic A. Sica, MD, and Douglas M. Landwehr, MD, *Richmond*.

Massive pleural effusion (PI ef) is an uncommon complication of CAPD. Though the etiology is unclear, mechanisms cited have included both macroscopic and/or microscopic defects in the diaphragm (D). Alterations in lymphatic (L) transport have also been proposed since CAPD may well augment the D flow of L. In addition, an impediment to L removal within the PI space results from hypoventilation, which is caused by the mechanical effect of the large fluid volumes impinging on the D. The exact management of this entity remains controversial, prompting our description of a CAPD patient whose PI ef did not recur on intermittent peritoneal dialysis (IPD).

A 63-year-old female developed a right PI ef 3 months after Tenckhoff catheter placement for CAPD. This persisted despite more than adequate volume removal. Thoracentesis, at which time the PI space was tapped dry, revealed a transudate with an elevated glucose concentration, suggesting transport of the hypertonic dialysate into the PI space. Radioopaque dye introduced into the abdomen by way of the Tenckhoff catheter did not appear in the PI space. This excluded a macroscopic defect in the D, although a microscopic defect could not be totally ruled out. PI fluid reaccumulated when the patient was restarted on CAPD. After 1 month of hemodialysis, the patient was placed on IPD. The ef has not recurred in 12 months of followup. The continuous presence of fluid in the abdomen during CAPD may lead to PI ef, whereas IPD will not. This may reflect the fact that IPD



allows for healing of D defects or that, since it is intermittent, L flow is not increased throughout the day. This is the second report in which the limited dwell times of IPD have resolved the PL of CAPD.

**High Prevalence of Renal Artery Stenosis in Patients with Hypertension Undergoing Cardiac Catheterization.** George Vetrovec, MD, Michael J. Cowley, MD, Douglas M. Landwehr, MD, and Virginia E. Parker, FNP, *Richmond*.

To assess the frequency of renal artery stenosis (RAS) in patients with hypertension (HBP) and/or renal insufficiency (RI) undergoing coronary angiography, screening abdominal aortography was performed in 118 patients with a history of HBP and/or RI. Single plane cine abdominal angiography was performed immediately following routine coronary angiography utilizing 20-40 ml of 76% renografin. Abdominal angiography was adequate for interpretation in 116 of 118 (98%) patients. One hundred and two patients had HBP 2 had RI (creatinine 1.5 mgs) and 12 patients had both HBP and RI (HBP/RI). RAS (>50%) was present in 26 of 116 patients (23%); 18 of 102 (18%) HBP patients, 1 of 2 RI patients and 8 of 12 (67%) HBP/RI patients. Significant RAS was present in 22 of 73 (30%) patients with coronary artery disease (CAD) while 4 of 43 (9%) patients without CAD had significant RAS. Associated decreases in kidney size were seen unilaterally in 3 patients while 2 patients had bilaterally decreased kidney size with associated bilateral sign RAS. In conclusion: 1) Significant RAS was identified in 30% of patients with HBP and/or RI suggesting that more vigorous consideration of RAS may be appropriate in HBP patients with CAD. 2) Abdominal angiography in conjunction with coronary angiography appears to be a useful screening test for RAS in patients with HBP and/or RI.

**Detection of Aortic Root Dissection by Two-Dimensional Echocardiography.** Jerome E. Granato, MD, Paul Dee, MD, George A. Beller, MD, and Robert S. Gibson, MD, *Charlottesville*.

Prognosis in aortic root dissection (ARD) is dependent on early diagnosis. Accordingly, we sought to determine the feasibility and predictive value of two-dimensional echo (2DE) in detecting DeBakey Type I or II ARD. Between November 1978 and June 1983, 10,385 patients underwent 2DE examination at our institution and 41 (0.4%) were performed because of suspected ARD. All studies were obtained at the bedside and in 36 patients (88%)

sufficient clinical and echo data was available for review. Standard 2DE criteria for ARD were employed. All 7 patients with ARD subsequently confirmed by arteriography or postmortem examination were identified. In 7 patients, a double lumen was visualized and 2 showed an oscillating intimal flap. Four additional cases proved to be false-positives by arteriography, computerized tomography or clinical course and 25 patients were correctly identified as negative for ARD, confirmed by similar criteria. These data indicate a 100% sensitivity and 86% specificity for ARD by 2DE with a positive/negative predictive value of 64% and 100% respectively. Thus 2DE appears to provide useful noninvasive information in patients with suspected ARD. Because it can be performed at the bedside, 2DE offers a capability to provide early diagnosis of this condition.

**Continuous Wave Doppler for Evaluating Adult Patients with Suspected Aortic and Mitral Valvular Stenosis.** Julia Y. Wen, MD, *Charlottesville*, R. Brad Stamm, MD, *Santa Fe, New Mexico*, and Robert S. Gibson, MD, *Charlottesville*.

The ability of two-dimensional echo (2DE) to reliably predict the hemodynamic severity of calcific aortic stenosis (AS) and mitral valve restenosis post-commissurotomy has been questioned. Because of this, we undertook a study to determine if a commercially available continuous wave doppler (CWD) could quantify the gradient of AS and accurately estimate valve area in mitral stenosis (MS). 76 patients had CWD within 24 hours prior to diagnostic cardiac catheterization; in 40 patients with suspected AS, maximum aortic gradient was calculated using the modified Bernoulli equation, and in 36 patients with clinical MS the valve area was derived from the diastolic pressure half-time equation of Hatle et al. The CWD gradient closely approximated cath in the 40 patients with AS; over a wide range of pressure gradients up to 160 mmHg, the  $R = .938$ ,  $p < .001$ . Importantly, CWD correctly distinguished all 19 patients with gradients <50 mmHg and 20/21 (95%) patients with larger gradients; only one patient with significant AS was underestimated by CWD (43 vs 76 mmHG). The correlation between mitral valve area by CWD and cath was also good;  $R = .883$ ,  $p < .001$ , and was of value in 5 of 7 patients post-commissurotomy with difficult mitral valves to evaluate by 2DE. Thus, our data suggests that CWD can accurately measure valve gradients and areas and these noninvasive measurements add considerable information to standard 2DE.

**Pulmonary Edema Complicating Treatment of Cardiac Tamponade.** Vaughn R. Barnick, MD, *Salem*, and Marta Sayers, MD, *Roanoke*.

Pulmonary edema following pericardiocentesis for cardiac tamponade has been reported only once previously. We report a case in a patient without pre-existing cardiopulmonary disease.

A 36-year-old black female was admitted with cardiac tamponade. Emergency pericardiocentesis was performed. 200 ml of pericardial fluid were removed, resulting in marked hemodynamic improvement. Recurrent tamponade necessitated direct surgical drainage for diagnostic and therapeutic purposes. Removal of 500 ml of fluid resulted in hemodynamic improvement followed by acute onset of pulmonary edema. No primary cardiopulmonary dysfunction could be demonstrated which may have been responsible for the onset of pulmonary edema. We postulate that a rapid decrease of the intrapericardial pressure below the right atrial pressure can result in a preload/afterload mismatch secondary to an acute elevation of preload combined with an elevated systemic vascular resistance. This may be avoided by limiting initial aspiration of pericardial fluid to that volume which corresponds with resolution of pulsus paradoxus and adequate hemodynamic improvement. Additional fluid should be removed slowly via an indwelling catheter.

**Infected Prosthetic Joint: Does Early Removal Improve Outcome?** Charles Durand, MD, and Stuart Rosenberg, MD, *Charlottesville*.

Despite anecdotal reports recommending early extirpation, the influence of prompt resection of an infected prosthetic joint on successful treatment of the infection has not been systematically examined. Charts of all patients seen at the University of Virginia Hospital in 1971-1981 with a discharge diagnosis of prosthetic joint infection (PJI) were reviewed retrospectively. The interval from PJI diagnosis to removal of the prosthesis and the occurrence of distant infections (endocarditis, bacteremia, meningitis, visceral abscess, or infection of a second joint) were noted. Early removal was defined as prosthesis removal within 3 months of PJI diagnosis. All others were classified as late removal. For each symptom (drainage, local pain), resolution of the symptom beginning within 3 months of prosthesis removal and lasting at least 12 months, defined treatment success. We identified 26 cases of PJI with 65 patient-years of followup (range 1-107 months, median 1.9 years). There were no deaths. Both groups had a high failure rate. Data fail to

show superiority of early removal. In selected patients with PJI, high operative risk from associated

Group	Patients	Distant Infections	Treatment Success	
			Drainage	Pain
Early	12	1	50%	11%
Late	14	1	50%	17%

illness or poor rehabilitation potential of available surgical salvage procedures could argue for delay in removal of the prosthesis. For such patients, the morbidity and mortality may be less with retention of the infected prosthesis.

**Isolated Ultrafiltration in Acute and Chronic Renal Failure.** Howard M. Lifland, MD, and Jorge Roman, MD, *Salem*.

At our institution, we have employed isolated ultrafiltration using an ultrafiltration device in the treatment of acute renal failure with fluid overload and for continuous slow ultrafiltration. This technique has also been utilized in end stage renal disease with an acute insult (e.g., hypotension, GI bleed) and/or acute volume overload. We present case examples of the use of this procedure in both acute isolated ultrafiltration and continuous slow ultrafiltration. A flow chart illustrating the use of acute isolated ultrafiltration follows:

Time (Hrs)	BP	Fluid Bal.	pO <sub>2</sub>	BUN/Cr.
0	90/40	0 ml	90	52/4.6
3	90/48	-1520 ml	100	81/5.6
5	101/60	-2220 ml	95	75/5.3

This method minimizes hypotension and hypoxia and also requires less anti-coagulation than standard hemodialysis. There is also a larger clearance of middle molecules than is achieved with hemodialysis. Ultrafiltration is comparable to hemodialysis in terms of control of azotemia with maintenance of stable osmolality and chemistries, thus eliminating large osmotic swings. The large volumes which can be removed make essential fluid replacement (TPN, pressors, alkali) less problematic, an obvious advantage in critically ill patients with precarious volume status. We believe these techniques have great potential for use in the care of unstable renal failure patients.

**Pseudoephedrine Accumulation in End Stage Renal Disease.** Nancy Radtke, MD, Domenic A. Sica, MD, Susan Glocheski, MD, and Antonia M. Harford, MD, *Richmond*.

Drug-prescribing patterns for patients with renal failure (RF) result in an increased prevalence of



adverse drug reactions. Over-the-counter drugs mistakenly viewed as benign are also capable of leading to adverse effects in RF. We report a case in which pseudoephedrine (P), a commonly utilized nasal decongestant, accumulated systemically in a patient undergoing maintenance hemodialysis (HD), who developed a pattern of neurologic symptoms commonly attributed to uremia.

The patient was a 64-year-old male with an extensive cardiovascular history. Following cardiac catheterization, acute RF developed, leading to the initiation of HD in January 1983. He became markedly depressed and developed myoclonic jerks despite more than adequate HD (12 hours/week). On 1-18 P 60 mg qid was started for nasal stuffiness and was continued through 2-1. 48 hours after P was discontinued, its plasma level as determined by radioimmunoassay was 1425 ng/ml (normal 100 ng/ml). HD clearance as measured by hourly arterial-venous sampling was significant (74 cc/min). Despite this, extensive volume of distribution for this drug ultimately limited quantitative dialysance of P. The neurologic symptoms abated during February. We cannot attribute with absolute certainty the neurologic symptoms to the persistent elevation of P or the resolution of these same symptoms to the decline in drug levels. However, this case underscores the importance of careful drug prescription in RF, a condition in which any drug ingested may accumulate with the potential for toxicity.

**Sleep Apnea Is Common in Active Acromegaly.** T. B. Hart, MD, S. K. Radow, MD, K. R. Cooper, MD, W. G. Blackard, MD, and H. St. G. Tucker, MD, *Richmond*.

Previous case reports have demonstrated an association between acromegaly and the sleep apnea (SA) syndrome, but the frequency of this association is unknown. Some of the reported patients had central SA, raising the possibility that acromegaly caused a defect in respiratory drive. We determined the prevalence of SA and hypercapneic ventilatory response (HCVR) in 21 patients with a history of acromegaly. We separated them into two groups based on serum human growth hormone (HGH) concentrations. Ten patients had active acromegaly (mean HGH 83.1, range 16.4-148), while 11 patients had inactive acromegaly (mean HGH 3.2, range 0.7-6.4). HCVR was measured by the rebreathing technique of Read, and SA was evaluated by polysomnography. 40% (4/10) of the patients with active acromegaly had SA. Three of the patients with SA had the purely obstructive type, and one had the mixed central and obstructive type. HCVR was

normal in all 20 patients tested, and was not influenced by the presence of SA nor by the level of HGH.

We conclude that SA is common in patients with acromegaly and infer that successful treatment of acromegaly also results in disappearance of SA. Specific therapy for SA in patients with acromegaly may not be needed if acromegaly can be successfully treated and symptoms of SA are not severe.

**Hyperthyroidism Caused by an Occult Source of Thyroxine: Struma Ovarii.** Steven Prince, MD, Jim Hsiu, MD, Kasedul Hoq, MD, and Herschel Estep, MD, *Norfolk*.

Struma ovarii is an uncommon tumor of the ovary and one which causes hyperthyroidism is rarer still. A 69-year-old woman presented with both clinical (tachycardia, weight loss, lid lag, stare) and biochemical (elevated T<sub>4</sub>, T<sub>3</sub>, depressed TSH) evidence of thyrotoxicosis. The thyroid was not enlarged, however, and the initial RAI was low (8%, 24 hrs), the low uptake thought to be secondary to an IVP done one week previously. RAI uptakes performed at 2 and 4 months subsequently, however, remained low. Hypermetabolism was controlled with PTU, but the thyrotoxic state returned when this medication was discontinued periodically. A right adnexal mass discovered when she was examined under anesthesia during evaluation for menorrhagia proved to be an ovarian tumor composed mostly of thyroid tissue. It contained T<sub>3</sub> and T<sub>4</sub> in concentrations well in excess of that of peripheral blood. No parafollicular cells could be identified but an indirect immuno-peroxidase (IIP) stain (which identifies parafollicular cells in the cervical thyroid) was strongly positive in the follicular cells of the struma. Calcitonin content of the struma was less than that of another age-matched ovarian specimen. Hyperthyroidism disappeared without medication and RAI uptake returned to normal (25%, 24 hrs) following removal of the struma ovarii.

Functioning ectopic thyroid tissue should be considered when hyperthyroidism occurs without thyromegaly and with low cervical thyroid RAI uptake. Positive IIP staining in our case is a unique finding and suggests an APUD cell origin of the struma.

**Ectopic Corticotropin Releasing Factor Production in Small Cell Undifferentiated Carcinoma of the Prostate: Case Report.** Shalendra Kumar Varma, MD, and Robert M. Carey, MD, *Charlottesville*.

Ectopic production of peptides possessing corticotropin releasing factor-like activity has been described in bronchogenic, thyroid, colonic and pan-

creatic carcinomas producing ectopic ACTH. Isolated production of ectopic CRF has not been reported. We present a 56-year-old white male who expired because of diffusely metastatic small cell undifferentiated carcinoma of the prostate. He presented with hypernatremia, hypokalemia, metabolic alkalosis and hypercortisolemia. An elevation in 24-hour urinary excretion of 17-OH ketogenic steroids and urinary-free cortisol was documented. Low dose and high dose dexamethasone suppression tests failed to decrease the urinary-free cortisol excretion. ACTH level was 165 pg/ml. Postmortem examination revealed diffuse small cell carcinoma of the prostate involving the liver, pleura, lungs, bone marrow, adrenal gland and the pituitary-hypothalamic infundibulum. Analysis of hepatic tissue laden with tumor metastases by radioimmunoassays utilizing heterologous antibodies to ovine CRF revealed the presence of corticotropin releasing factor without concomitant presence of ACTH. Microscopic examination of the pituitary gland found it to be hyperplastic, presumably in response to CRF. We believe this to be the first reported case of ectopic corticotropin releasing factor production without concomitant ectopic ACTH secretion.

**Diabetes Insipidus Associated with Leukemia.** Bernard Ilson, MD, Domenic A. Sica, MD, Michael Eleff, MD, and William T. Dabney III, MD, *Richmond*.

Leukemia is a disease whose systemic involvement tends to be protean in nature. Despite the occurrence of perihypophyseal leukemic infiltrates in as many as 46% of acute leukemia patients, overt diabetes insipidus (DI) is a rare occurrence in this disease. Chronic myelogenous leukemia (CML) in particular rarely causes DI, with fewer than 4 cases currently cited in the world's literature. We have recently had the opportunity to evaluate a 54-year-old female with long-standing CML who developed central DI. This patient had a 5-year history of CML and had recently entered into a blast phase. On presentation, serum sodium was 154 meq/L with a urine S.G. of 1.005. Initial attempts at hydration resulted in the elaboration of large quantities of hypotonic urine. At that time, urinary osmolality was 168 mosm/kg, plasma osmolality 345 mosm/kg and plasma ADH was .2uU/ml (nl 0.3-2.0) with urine outputs of 600-700 cc/hr. Following 5 units of aqueous vasopressin, urinary osmolality increased to 473 (280% increase). Urinary output and serum tonicity normalized with the inception of therapy with intranasal desmopressin acetate (DDAVP). Workup included computerized tomography, neu-

rophypophyseal testing and lumbar puncture for malignant cells and was entirely negative. Subsequently, the patient was hospitalized with widespread pneumonia and expired. An autopsy was not obtained. Possible pathophysiologic mechanisms for this entity might include diffuse leukemic infiltration of the posterior pituitary or small vessel thrombosis in hypothalamic nuclei and the posterior pituitary.

**Renal Cell Carcinoma in End Stage Renal Disease.** Daniel Kebede, MD, and Jorge Roman, MD, *Salem*.

Relatively little attention has been focused on the fate of renal tissue once renal failure reaches its end stage. The development of renal cysts and enlargement of kidney size have been described in chronic hemodialysis patients. It has been reported that these cysts could be premalignant and renal cell carcinoma may arise from these cysts. We have found about 20 reports of de novo renal cell carcinoma in chronic hemodialysis patients. We present the case of a patient on long-term hemodialysis who developed renal cell carcinoma of the right kidney after 10 years on hemodialysis. The patient underwent a right nephrectomy and since then he is being followed with yearly CT scans of the abdomen with no evidence of the disease in the contralateral kidney. In a literature search, we have not found any followup strategy for chronic hemodialysis patients. It appears that these patients are at a high risk of developing renal cell carcinoma and the risk increases with the number of years on dialysis. Since in renal cell carcinoma early diagnosis and resection guarantee almost a 65% 5-year survival rate, we suggest an aggressive followup of these patients either with CT scan or sonography of the kidneys.

**A Scleroderma-Like Syndrome Associated with Gastric Adenocarcinoma.** David Maxwell, MD, *Norfolk*.

A syndrome of acute generalized cutaneous sclerosis not attributable to another dermatologic, endocrinologic or rheumatologic disorder which microscopically resembles scleroderma is described as a paraneoplastic syndrome of gastric adenocarcinoma in a 72-year-old woman. The face, neck, shoulders, chest, back and extremities were involved. There were strikingly abrupt lines separating normal and involved skin. No telangiectasias, ulcers or vesicles were present. Dysphagia was described with hypomotility of the lower esophagus. Chest X-ray and lung diffusion capacity were



normal. Circulating immune complexes were detected by Raji cell assay, ANA was 1:40 homogeneous pattern, muscle enzymes, renal function and cardiac exam were normal. Skin biopsy revealed increased dermal collagen, with entrapment of sweat glands and epidermal atrophy. Amyloid stains and immunofluorescence were negative. Capillary basement membranes were thickened on electron microscopy, suggesting repeated endothelial damage consistent with findings in progressive systemic sclerosis. There was no direct evidence for polymyositis, carcinoid syndrome, amyloidosis or fasciitis. There are 4 reported cases of paraneoplastic scleroderma; we believe this to be the first case associated with gastric adenocarcinoma.

**Systemic Fibrosis and Intracytoplasmic Crystalline Aggregates in a Chronic Lymphocytic Leukemia Patient.** John Wright, MD, Munsey Wheby, MD, Kuldeep Teja, MD, and Donald Innes, MD, *Charlottesville*.

A 66-year-old white male with chronic prolymphocytic leukemia and a biclonal gammopathy (IgM and IgG  $\lambda$ ) developed fever, pulmonary infiltrates, progressive congestive heart failure and abdominal pain 2 years after diagnosis and initiation of chemotherapy. Hematologic status at admission included WBC count 23,900, Hct 33.4 with massive splenomegaly but without lymphadenopathy. Despite aggressive evaluation (including lung biopsy, invasive hemodynamic monitoring), the patient developed progressive respiratory deterioration and cardiac failure and expired after 19 days. Postmortem examination revealed extensive epicardial mediastinal and retroperitoneal fibrosis that exceeded 2 cm thickness in several areas with no compression of internal organs. Light and electron microscopic examination revealed intracytoplasmic crystalline aggregates present in histiocytes as well as alveolar and renal tubular cells. Average crystal size was approximately  $1.5\mu \times 0.3\mu$ . The aggregates have been identified as immunoglobulin, based on staining with immunoperoxidase-labeled antibody specific for heavy chains of IgM and IgG as well as lambda light chains.

Intracytoplasmic crystalline aggregates of immunoglobulin have been described in peripheral leukocytes of chronic lymphocytic leukemia patients. Although decreased serum gammaglobulin levels were found in most reported cases, a monoclonal gammopathy was seen in a few patients. The present case seems to be the only one in which the immunoglobulin aggregates were associated with a biclonal gammopathy.

## Can You Diagnose This?

Answer to puzzle on page 90.

### DIAGNOSIS

Left atrial myxoma

### DISCUSSION

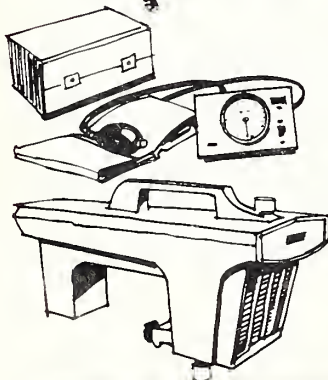
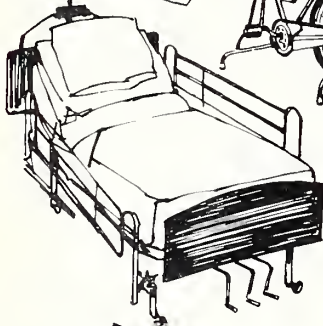
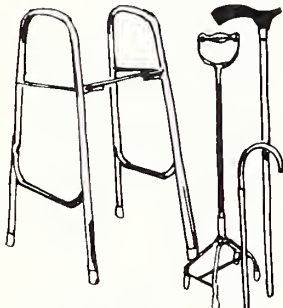
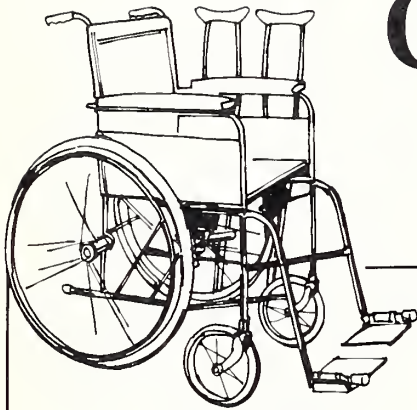
Left atrial myxomata, the commonest intracavitary tumors of the heart, usually arise on a pedicle from the interatrial septum and display varying degrees of mobility, depending on the length of their pedicles. The pedicle may be short, allowing little movement of the tumor, or it may be long, allowing the tumor to prolapse into the left ventricle during diastole and to be thrust back into the left atrium during systole.

The patient's cross-sectional echocardiogram (during diastole) reproduced on the previous page shows the typical features of a large prolapsing left atrial myxoma. A large well-circumscribed mass is seen arising in the left atrium and prolapsing through the mitral orifice into the left ventricle.

M-mode studies of patients with left atrial myxomata (not shown here) typically demonstrate a mass of echoes behind the mitral valve or in the left atrium. Although M-mode echocardiography has proven to be a valuable screening procedure in the detection of intracardiac tumors, the narrow ultrasound beam used in this technique provides only limited information about a tumor's spatial orientation within the heart. The cross-sectional technique, with its ability to produce a slice-like tomographic image of a portion of the heart, provides a substantial advantage over the M-mode examination by allowing a more precise evaluation of a tumor's size, shape and mobility. In addition, it is more sensitive than the M-mode for the detection of small, non-prolapsing or multiple tumors. In young patients without clinical evidence of coronary artery disease, the information obtained by cross-sectional echocardiography is often sufficiently complete and reliable to permit operative removal of the tumor without additional invasive studies.

It should be pointed out that although non-invasive echocardiography can reliably detect intracardiac masses, it cannot define their histology, and it may sometimes be difficult or even impossible to differentiate a left atrial myxoma from a large left atrial ball thrombus or other intracardiac neoplasm.

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# VIRGINIA MEDICAL

# EDITORIAL

## A Brochure to Help You Cope

**I**COMMEND to your attention the brochure bound into this issue at page 86, where it can be pulled out for your perusal. It is about diagnostic-related groups, or DRGs, the prospective payment system that is the latest in the long line of government-imposed health care plans. The brochure was developed for Virginia physicians by a subcommittee of The Medical Society of Virginia's Vanguard Committee composed of Dr. Joseph V. Puglise, chairman, Dr. Ira J. Green, Dr. William J. Hagood, Jr.,

and Dr. C. M. Kinloch Nelson. Dr. J. Hayden Hollingsworth is Vanguard chairman.

Almost every physician is aware of DRGs, but a relatively few are fully informed. It is essential that all physicians know the basic facts, at least insofar as such are available. I believe you will find in this brochure information both new and helpful.

The Editors are delighted that VIRGINIA MEDICAL can bring you this timely booklet.

EDWIN L. KENDIG, JR., MD

## Virginia's Trauma Centers

**A**N ESTIMATED 65 million injuries occurred in 1983 due to trauma. Of these, 164,000 were fatal. Accidents remain the third most common cause of death in the United States and the leading cause of death in those under 40 years of age. The overall cost, including wage losses, medical expenses, insurance, administrative costs and property damage, is thought to be about \$83.5 billion per year.

Both military and civilian experiences indicate that implementation of an integrated and organized trauma system can reduce death disability and health care costs. Effective response to a trauma

emergency must begin at the scene of the accident with well-trained emergency medical technicians. The trauma system must ensure that the emergency medical technicians transport the patient to a trauma center capable of continuing and expanding life-support activities begun at the scene. In a trauma center, skilled surgical personnel are available to provide immediate care of the trauma victim. Since the trauma center is not usually the nearest hospital in urban areas, the emergency medical technicians must bypass hospitals without expertise in trauma care, transporting the patient to the trauma center.

The importance of this pre-hospital triage deci-



sion has been demonstrated by recent clinical studies.<sup>1</sup> Preventable deaths were reduced from 73% to 9% when the accident victim was treated in a designated trauma center. If the trauma victim was inappropriately triaged to a hospital without this expertise in trauma care, the percentage of preventable deaths remained at 67%.

As authors Warren, Ward and Becker incisively describe elsewhere in this issue, prompt diagnosis and expeditious transfer to a trauma center is particularly important in patients with head injuries.<sup>2</sup> Treatment of these patients with intracranial monitoring and early operative intervention within four hours after injury can maintain brain function. A delay in treatment results in brain damage that cannot be salvaged.

In rural communities without trauma centers, interhospital transport of the critically ill patient to the trauma center is mandatory and becomes an integral part of the statewide trauma system. The mode of interhospital transport is an important consideration in developing a trauma system. When comparing the survival of two groups of patients with injuries of comparable severity, the survival of those transported by helicopter was greater than those transported by ground ambulance.<sup>3</sup>

Ensuring the delivery of the critically injured patient to the appropriate hospital requires that communities inventory, categorize and designate the capabilities of their hospitals in delivering trauma care. Cognizant of the importance of this regional approach to trauma care, Gov. John Dalton directed the Virginia Department of Health on August 7, 1981, to designate regional trauma centers. Hospitals designated as trauma centers must make the commitment detailed in the American College of Surgeons' trauma center criteria.<sup>4</sup> Through this commitment of resources, the desig-

nated trauma center can develop a responsive program that provides specialized care to the trauma victim.

The University of Virginia School of Medicine and the Medical College of Virginia/Virginia Commonwealth University have been designated as trauma centers. At each institution the designation brought a positive change in attitude toward the trauma patient by the hospital staff, especially surgical and emergency personnel but also hospital administrators, teachers, scientists and support personnel, and gave impetus to in-house reorganization that was necessary to develop a highly skilled multidisciplinary trauma service.

In addition to reducing death, disability and health care costs, trauma centers are serving as educational resources for training surgeons, nurses and other specialists in the care of the trauma victim. They are also playing key roles in implementing public educational programs focusing on accident prevention.

RICHARD F. EDLICH, MD

Department of Plastic Surgery  
University of Virginia School of Medicine  
Charlottesville VA 22908

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3. Baxt WM: Impact of a rotorcraft aeromedical emergency care service on trauma mortality. *JAMA* 249:3047-3051, 1983
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## Reflections on Prevention

**I** WONDERED, during the recent 40th reunion of my medical school class, what my classmates and I knew about preventive medicine at graduation and the start of our careers, and if we had changed our thoughts in the long time since then.

Most of the members of the Class of '43 served in the armed forces, where we must have been impressed by successes in the control of some of the infectious diseases that had been scourges in previous conflicts, and where the preventive duties of

medical officers required approaches not emphasized during our student and intern days.

Subsequent training and experience in the delivery of health care during a period of rapid improvement in immunization practices and knowledge of proper nutrition, with beginning success in accident control and concern for pervasive social and environmental problems, showed me that trying to help people to stay well is a worthwhile use of my efforts.

### THANK YOU!

The Editors greatly appreciate the special tasks of review and consultation performed during 1983 by the following:

George W. Vetrovec, MD  
Lockhart B. McGuire, MD  
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Robert E. Petres, MD  
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We pediatricians can have pride in our track record. I must tell the medical students that I cannot show them patients who have typhoid fever, diphtheria, poliomyelitis and tetanus, all of which I have seen. Measles, whooping cough and tuberculosis are still with us but greatly reduced in incidence. Even iron deficiency anemia of nutritional origin seems to be less common at the University of Virginia Medical Center. It is not easy for me, and it must be many times more difficult for the students, to cope with the fact that the world is full of people with severe malnutrition and infectious diseases, both potentially preventable. There is a challenge to be concerned about the anticipation and recognition of maladies that no longer occur commonly yet can be catastrophic in conditions of communal disaster.

The turnaround by the conquest of rubella, after I had been taught that it was an unimportant disease that nevertheless caused an unbelievable epidemic of devastating congenital defects, followed by the worldwide eradication of smallpox, transiently made me imagine that I was truly practicing in the age of medical miracles, especially when I combined these thoughts with what antibacterial medicines would do for my sick patients. Oh, I should

have known better! Look what happened with the penicillin-resistant staphylococcus and the reactions to the "wonder drugs".

Nevertheless, I found myself thinking that we needed new fields to conquer, and that common, chronic, systemic disease conditions, many beginning in childhood, should be approached by asking why they occur, what causes them and what can be done to prevent them. The nascence of true, clinical, genetic services fell into line with these considerations. Improved understanding of the hereditary components of familial diseases, overtly accepted by doctors, who must help their patients to also accept these possibilities, through more and better use of education and guidance, make it seem logical that we will grow to control the devastations of cardiovascular disease, diabetes mellitus, obesity, cancer, arthritis, some mental illnesses, and learning disabilities. We can learn to prevent venereal diseases. I believe that these things will happen.

Idealism? Of course. Visionary? Pray tell me, why not be? However, reality dictates that I must recognize the practical things involved, such as the mind-boggling cost of the necessary research, and the more and more complex and expensive patient care. These will of necessity directly relate to what now is called cost-effectiveness: Will there be a finite boundary to what we can spend? And can we afford to continue the propagation of those who pass along defects? What will we do about gene manipulation? Will it affect you or your progeny?

"Health promotion" is the current term for many of the preventive efforts of the caring, personal physician that often is at variance in "a highly scientific, technologic and curative profession in parallel with that of a technologic society."<sup>1</sup> There is a fascinating, current awakening of an alliance between ancient, herbal medicine and traditional medicine that in some parts of man's world will offer practical benefits to those prepared to accept them. The preservation of health and the control of disease and injury must be essential, basic concepts in medical education and training.

And we will still have accidents, all of the abuses, familial disruption, emotional disorders, complications of pregnancy (including prematurity), problems of aging, and war and pestilence.

I believe that the art and science of medicine will continue to be demanding of us for a long time.

ARMISTEAD P. BOOKER, MD

1. Totten MA: Physician's role in health promotion (letter). *N Engl J Med* 1983;308:1424-1425



# VIRGINIA MEDICAL OBITUARY

## **Rachel W. Gann, MD**

Dr. Rachel Weems Gann, former director of physical medicine and rehabilitation at the Woodrow Wilson Rehabilitation Center in Fishersville, died October 31, 1983. She was 87 years old.

She was a native of Ames, Iowa, and moved to Virginia in 1904. Twenty years later she received her medical degree from the Medical College of Virginia, one of the first women to do so. From 1925 to 1944 she was resident physician and professor of health education at Madison College, now James Madison University, in Harrisonburg. She then trained for five years at the D. T. Watson School of Psychiatrics in Pittsburgh, after which she returned to MCV for a three-year term as assistant professor of clinical physical education. She established a practice in Ashland and remained there until 1956, when she was appointed to the Fishersville post, where she served for 13 years.

A member of the Albemarle County Medical Society and The Medical Society of Virginia for 58 years, Dr. Gann was also a member of the American Academy of Physical Medicine and Rehabilitation.

## **Frank R. Hopkins, MD**

Dr. Frank Read Hopkins, Lynchburg pediatrician and past president of the Lynchburg Academy of Medicine, died September 19, 1983, after a long illness. He was 80 years old.

A native of Hot Springs, Virginia, Dr. Hopkins was graduated from the University of Pennsylvania and received his medical degree from the University of Virginia in 1926. He interned at the Monroe County Hospital, Rochester, New York, and trained also at Louisville, Kentucky, General Hospital and Childrens Hospital Medical Center, Boston. He established his practice in Lynchburg in 1931, leaving only during World War II, when he was assigned to the Pacific, serving the US Navy as senior medical officer on the *USS Mount McKinley*.

A past president of the Virginia Pediatric Society, Dr. Hopkins was also a member of the American Academy of Pediatrics and American Medical Association. He came to membership in The Medical

Society of Virginia over 50 years ago, through the Lynchburg Academy of Medicine.

## **Benedict Nagler, MD**

Dr. Benedict Nagler, Lynchburg psychiatrist and former director of the Lynchburg Training School and Hospital, now Central Virginia Training Center, died December 16, 1983, at Virginia Baptist Hospital. He was 83 years old.

Born in Czernowitz, Germany, Dr. Nagler was graduated from the University of Hamburg, Germany, Medical School in 1925. He came to the United States ten years later and settled in Newark, New Jersey, where he conducted a neurology practice until 1943. He then joined the US Army and was chief of psychiatry and neurology at Cushing General Hospital in Framingham, Massachusetts. At the end of World War II he was appointed chief of psychiatric and neurological services at the McGuire Veterans Administration hospital in Richmond. In 1953 he moved to Washington, DC, and worked for four years in the Veterans Administration hospital there. He was appointed chief of the training center in Lynchburg in 1957 and held the post until his retirement in 1973, when he continued to practice on a limited basis as a consultant for court attorneys.

Dr. Nagler was a member of the American Academy of Neurology, American Psychiatric Association, and the Lynchburg Academy of Medicine. He had been a member of The Medical Society of Virginia since 1959.

## **Herman F. Oppleman, MD**

Dr. Herman F. Oppleman, for 42 years a general practitioner in Richmond, died October 26, 1983, at the age of 75.

A Richmond native, Dr. Oppleman was graduated from the University of Richmond and the Medical College of Virginia. He received his medical degree in 1930 at the age of 22, making him one of the youngest graduates in the history of MCV. He served the Army Medical Corps in the Pacific during World War II, then resumed his practice in

Richmond and continued to make house calls until his retirement in 1973. He was also the physician for the Virginia State Athletic Commission.

Dr. Oppleman's membership in the Richmond Academy of Medicine and The Medical Society of Virginia spanned 50 years.

### **A. Randolph Garnett, MD**

Dr. Alfred Randolph Garnett, Norfolk obstetrician and gynecologist, died November 21, 1983, at age 69.

A native of Norfolk, Dr. Garnett was graduated from the University of Virginia in 1935 and from the university's medical school in 1940. He received training at Garfield Memorial Hospital, Washington, DC, and later at Women's Hospital of the State of New York, New York City. He served as flight surgeon for the US Air Force during World War II and was based in Shrewsbury, England. He returned to Norfolk to begin his 35-year-long practice there, and he continued to conduct a limited practice after his retirement in 1981.

Dr. Garnett was a past president of the Virginia Society of Obstetrics and Gynecology and a member of the The Medical Society of Virginia, Norfolk Academy of Medicine and American College of Obstetrics and Gynecology.

A son, Dr. Alfred Randolph Garnett, Jr., practices medicine in Richmond.

### **James H. Powell, MD**

Dr. James H. Powell, longtime Petersburg surgeon and emergency room physician, died October 26, 1983, at the age of 65.

A Petersburg native, Dr. Powell earned his medical degree in 1943 at the University of Pennsylvania Medical School, Philadelphia, and trained at the Medical College of Virginia Hospitals, Richmond. He served in the US Army from 1944 to 1946, then returned to Petersburg to establish his practice. At the time of his death, Dr. Powell was in his 15th year as emergency room physician at Petersburg General Hospital.

He was a member of The Medical Society of Virginia, Southside Virginia Medical Society, American Society of Abdominal Surgeons and American College of Emergency Physicians.

## **Memoir of Charles D. Smith 1907-1982**

*By Reuben D. Knopf, MD, and  
John A. Martin, MD*

Born in Texas in 1907, Dr. Charles D. Smith died July 20, 1982, in Roanoke, Virginia. He received his bachelor's degree from Texas A & M and his MD from Baylor University. His residency in radiology was at Johns Hopkins University.

He began his practice in Richmond but soon moved to Roanoke. During World War II he served as a colonel and chief radiologist with the 45th Army General Hospital in North Africa and Italy. Returning to Roanoke after the war, he resumed his duties as chief of radiology at Roanoke Memorial Hospitals, where he later was chief of nuclear medicine and twice served as chief of staff.

A clinical associate professor at the University of Virginia School of Medicine, he was a founder of the Eastern Radiologic Society and was its president in 1963. He also served as president of the Virginia Chapter, American College of Radiology, and was a trustee of the American Registry of Radiologic Technologists.

A phrase by Edmund Burke seems appropriate for a radiologist: *What shadows we are, and what shadows we pursue*. Dr. Smith spent his adult life pursuing shadows, not the shadows of life, but the shadows of disease and death. In the darkness of a radiograph or the blackness of the dots of a nuclear image, he tirelessly strove for better technique and methods to facilitate pursuit of disease.

After more than 20 years in radiology, he undertook the challenge of a new field—"medical isotopes" as it was then called. He continued this pursuit in nuclear medicine with enthusiasm and vigor until a short time before his death. His endeavor took him to an appointment as an examiner in nuclear medicine for the American Board of Radiology. He established one of only two schools in the State of Virginia for the training of nuclear medicine technologists. As radiology grew, Dr. Smith grew with it. His inquisitive mind never dulled.

Although Dr. Smith spent his professional life in pursuit of shadows, the rest of his life was not in darkness but in the light of a deep love and devotion for his wife, Genevieve, who predeceased him by eight months, and his three sons, Doug, Stewart, and Kent. His leisure was in the brightness of the golf course or at his lake home. His pleasures included the warmth of many friends and sharing



his bourbon and conversation with them. His interest in correct English usage was legendary and the wit of H. L. Mencken never failed to amuse him.

He worked hard and long to improve medical practice, particularly at Roanoke Memorial Hospitals and also throughout the Roanoke Valley.

In life, Dr. Smith cast a long shadow. His pursuits are ended. May he rest in peace.

## Memoir of George Kriegman 1917-1982

*By William W. McDonough, MD,  
L. Martin Harris, MD, and Sherman Master, MD*

We were saddened on November 7, 1982, by the death of our friend and colleague, Dr. George Kriegman, whose career in Virginia spanned almost four decades.

Born September 14, 1917, in Chicago, Dr. Kriegman obtained a BA and an MS from the University of Illinois, graduating in medicine there in 1943. A rotating internship and psychiatry residency were completed by 1945 at St. Elizabeth's Hospital in Washington, DC. While a captain in the Army, he became the director of the Mental Hygiene Clinic for trainees at what is now Fort Lee. By the time of his discharge in 1947, he and his wife Lois, a clinical psychologist, had decided that Richmond would be an interesting place to practice and a good place to bring up their children. Upon completion of his analytic training at the Washington Psychoanalytic Institute in 1951, he became the first privately practicing psychoanalyst in the State of Virginia.

The education and training of all who were concerned with mental and emotional suffering was an abiding concern with Dr. Kriegman. He was first appointed to the clinical faculty at the Medical College of Virginia in 1950 and at the time of his death was clinical professor of psychiatry and chairman of the Clinical Faculty Committee of the Department of Psychiatry. At various times during his long and active career, he was lecturer in psychiatry at the University of Virginia, the School of Social Work at the Richmond Professional Institute, and the School of Nursing at MCV. As a consultant, he was well known by the Family and Children's Services of Richmond, the Veteran's Administration Hospital in Roanoke, the Richmond Children's Aid Society, the Child Care Bureau of the Virginia State Department of Public Welfare, and Westbrook Hospital.

All who trained with him will remember the care

and devotion with which he dedicated himself to both the patients and their therapists. His lectures, seminars and supervision became an integral part of the training program in the Department of Psychiatry at the Medical College of Virginia, and he was named Outstanding Professor by the psychiatry residents in 1981. Most will remember his management of the 22nd Annual Stonebrenner Lecture Series: Psychiatry and Medical Practice, and his subsequent guest editorship of the MCV Quarterly in 1969 as significant contributions to the physicians of Richmond.

Activity in professional societies was a hallmark of Dr. Kriegman's career. In addition to his membership in the Richmond Academy of Medicine and The Medical Society of Virginia, he was a life fellow of the American Psychiatric Association and the Neuropsychiatric Society of Virginia. He was a member of the American Psychoanalytic Association, the Academy of Psychoanalysis, the Washington Psychoanalytic Society, co-founder and past president of the Virginia Psychoanalytic Study Group, co-founder and first president of the Virginia Psychoanalytic Society, and founding president of the Richmond Psychiatric Society. His untiring devotion to these latter groups was largely responsible for their establishment and eventual stabilization as permanent societies in our community. Many of the organizational meetings were held in his home, lasting far into the night.

Dr. Kriegman was an avid gardener and table tennis player and he shared these skills with everyone who was interested. The Kriegman home was a center of hospitality for both work and play. He was the warm and cordial host of many pleasant social evenings, and even conducted business meetings around a bountiful dinner table, making the work less dour. Evenings frequently ended with a quick round of table tennis and a generous supply of vegetables from his garden to take home.

A caring but strict therapist, Dr. Kriegman was firmly allied with his patients and against their pathology. A former patient wrote of him that when you were terrified of drowning, Dr. Kriegman would teach you to swim rather than throw you a life preserver.

Dr. Kriegman will be missed; the impact of his mental acuity and human warmth on his family, students, colleagues, friends, community, state and his science will constitute an important and enduring memorial.

# WHO'S WHO

"A man of whom we are all very fond."

So **Dr. Harold L. Williams** wound up his encomium of **Dr. F. Ashton Carmines, Jr.**, when he presented the 1983 Physician Award for Community Service to the Newport News orthopedist. The presentation took place at the banquet that culminated The Medical Society of Virginia's annual meeting at the Homestead.

It would be hard not to like "Pete" Carmines, in whose unfailing affability the eyes of a mischievous boy still twinkle, ready for fun. There's nothing boyish, however, about Dr. Carmines' accom-

plishments, for which he was given the award.

A veteran leader, he has been President of The Medical Society of Virginia, the Newport News Medical Society and the Virginia Orthopedic Society. Riverside Hospital has known him as chief of surgery; Blue Cross Blue Shield of Virginia as a director; the Eastern Virginia Medical Authority as a commissioner; and the Warwick Kiwanis Club as director of its orthopedic clinic.

A man of deep religiosity, Dr. Carmines helped found North Riverside Baptist Church in Newport News and has served that congregation as trustee, treasurer, teacher, deacon and president of the Brotherhood. On missions combining his faith and his profession, he has travelled the world. For Orthopedics Overseas, a division of Care-Medico, he contributed tours of duty in Jordan, in 1962; in Hong Kong, 1964; and in Nigeria, 1967. Under the auspices of the foreign missions board of the Southern Baptist Convention, he spent a month at Mati Baptist Hospital in the Philippines in 1979.

The Kiwanians have given him their distinguished service award for his work with crippled children; for the Peninsula Youth Challenge he has worked with adolescents plagued by drug and alcohol problems; and the elderly of the Virginia Baptist Home have been buoyed by his Bible teaching.

A thoroughgoing Virginian, Dr. Carmines was born in York County, educated at the College of William and Mary and the Medical

◀ His community the world:  
Winner Carmines

College of Virginia. After a two-year stint with the US Army in 1944-1946, he trained and fulfilled a fellowship in Richmond and Newport News, then set up his practice in the waterside city that gives him plenty of places to go fishing.

Dr. Carmines is the 21st Medical Society of Virginia member to receive the award, which was established in the early 1960s by the A. H. Robins Company and has continued ever since under the pharmaceutical firm's sponsorship.

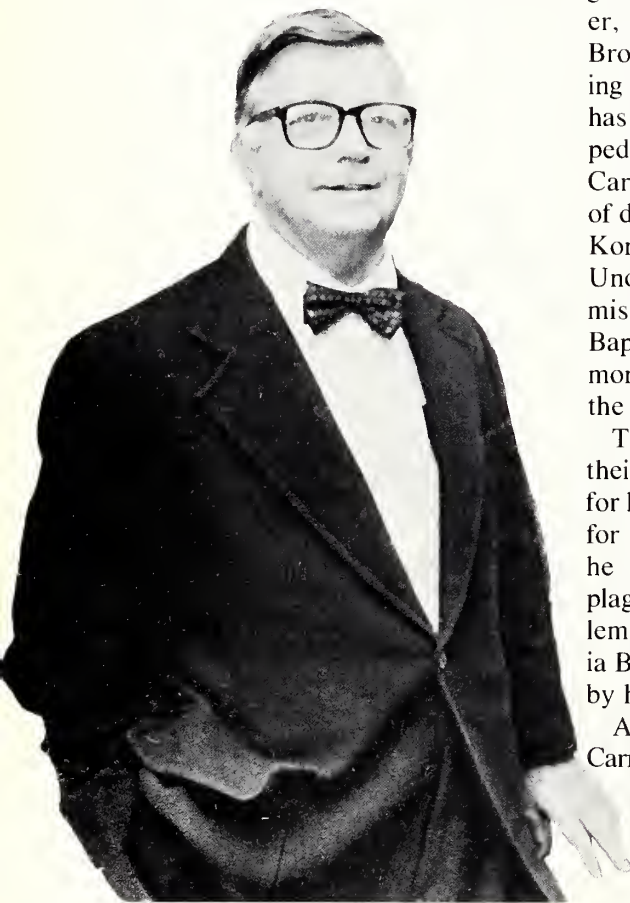
The leadership of Virginians ebbed and flowed at the annual meeting of the Southern Medical Association last November in Baltimore. **Dr. M. Pinson Neal, Jr.**, Richmond, departed the associa-



**President Reardon**

tion's presidency, while **Mrs. William J. Reardon, McLean**, came in as president of the SMA Auxiliary.

Well known for her leadership in local, state and national auxiliaries, Kay Reardon has been president of the Medical Society of Virginia Auxiliary, national chairman of the American Medical Association's Education Research Foundation, and vice president for the AMA Auxiliary's Eastern Region. Beginning in 1972, when she was elected a vice councilor, Mrs. Reardon has served the SMA Auxiliary in offices of increasing responsibility.







New members of the 50-Year Club gathered for the photograph above when The Medical Society of Virginia met in November at the Homestead. In the front row, left to right, are Dr. William M. Bickers, Richmond; Dr. Mary E. Johnston,

Tazewell; Dr. Harold E. Gillespie, Richmond; and Dr. Samuel Francis Driver, Roanoke. Second row, from left, Dr. Paul Hogg, Newport News; Dr. William Penn Frazer, Purcellville; and Dr. Andrew Dishart Shapiro, Roanoke. Third row, Dr. Rus-

sell V. Buxton, Newport News, left, and Dr. John Adrian Sims, Alexandria. On the stairs above them are, in ascending order, Dr. Roger Conant, Radford; Dr. J. Hamilton Allan, Charlottesville, and Dr. Raphael H. Greenstein, Salem.



# VIRGINIA MEDICAL CLASSIFIED

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**PHYSICIAN** wanted to associate with another doctor in established inner city practice in Richmond. Immediate opening. Mail replies to Alfred Pedynekowski, MD, 1208 Hull St., Richmond VA 23224.

**EMERGENCY MEDICINE**—Full-time positions available in four emergency departments located in eastern, central and western Virginia. Competitive income and professional liability insurance provided. Reimbursement for ACLS and ATLS training, CME tuition, ACEP dues. For details respond in confidence to: Katie Sherrill, Spectrum Emergency Care, Inc., 1111 N. Westshore Blvd., Suite 211, Tampa FL 33607, (813) 870-2356.

**HEALTH DIRECTOR** sought for Norfolk, Virginia, Dept. of Public Health. Oversee administration of environmental, preventive, diagnostic, therapeutic, educational, research programs at seven separate sites. Manage budget of 8½ million dollars. MPH, extensive background in public health required, or equivalent combination of education and experience. Must be Virginia licensed MD or eligible. Excellent benefits include liberal leave policy, paid health and malpractice insurance, retirement plan. Salary negotiable. Opportunity to teach at local medical school, college, university. Norfolk is a seaport with historical, cultural, recreational attractions. Resumes must be received by 5 PM, March 1, 1984. Specify job #1648, to Virginia Dept. of Health, Rm. 110, Madison Bldg., 109 Governor St., Richmond VA 23219. For more information, contact William B. Crawford, MD, Regional Medical Director, (804) 460-5314. Equal Opportunity Employer.



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*Cover photograph by Cyane B. Lowden*



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## Senate seat suits Clancy Holland

AS A PAGE in the Virginia senate 40 years ago, 14-year-old Clarence Adrian Holland watched the late Dr. James D. Hagood legislate effectively as a senator from Halifax County while continuing his general practice of medicine.

Today "Clancy" Holland has an established general practice of his own and is serving his first term as senator from Virginia Beach.

He joins there Dr. John C. Buchanan, senator from Wise County. Both are Democrats, but only Dr. Holland is a member of The Medical Society of Virginia, and its Vanguard Committee. His boyhood role model, Dr. Hagood, was MSV President in 1957.

"Working as a page whetted my appetite for state government," Dr. Holland remembers.

But, he adds, it was his father's long career as a legislator that really got him started: The late Shirley T. Holland, an Isle of Wight banker, served for 20 years in Virginia's House of Delegates. One of Clancy Holland's two brothers, Richard J. Holland, followed his father both in banking and in politics; he is the Democratic state senator from Isle of Wight. The third brother is Dr. William E. Holland, Richmond cardiologist. Not related to the three brothers is another current state senator, Edward M. Holland, D-Arlington.

There's no worry about who's minding the patients while Dr. Hol-

land's at the legislature, because these three associates endorse his role as a legislator and are filling in for him: Dr. George S. Wong, Dr. William B. Warden and Dr. William T. Johnson. That's mighty nice of them, because being a part-time legislator suits Clancy Holland just fine, he told a reporter.

"The most satisfying aspect of government is hearing people's ideas on how the government should be run, then making a decision that is in the best interest of all the people, not just the special interest groups," he observed.

"I believe we are put on this earth to help each other, and I like to feel I'm doing a service."

The new senator is no stranger to public office. He was for 12 years a member of the city council of Virginia Beach and for two of those years,



Tim Wright

**Dr. Clarence A. Holland (right), new state senator, and elder statesman Sen. Edward E. Willey at the party described in this issue on page 126.**



# NEWS BUREAU

1976 to 1978, was the city's mayor. He's proud of the work he and his colleagues did there.

"We kept a tight rein on the size of the government; we instituted a tourist and industrial development department; and we managed the city's tremendous growth by putting a lot of demands on builders, like making them run sewer lines for new residential developments.

"We gave strong support to education, including Eastern Virginia Medical School, and we developed excellent volunteer emergency medical services."

As for his Senate committee appointments, Dr. Holland had hoped to be named to Health and Education, but that committee was full, and he landed in these three: Agriculture and Natural Resources; and Social Services and Privileges and Elections. The last appointment is a rare one for a freshman senator; with it, Dr. Holland hopes to work toward making registration more accessible—but he's not convinced that postcard registration, as recommended by some, is the answer.

Strong support for education is a continuing legislative priority with Clancy Holland. An advocate of the Virginia Education Association's "meet and confer" proposal, he points out that the Virginia Beach school board has been meeting and conferring with its teachers for years. He wants to see Virginia teachers' base salaries at a level "commensurate with their education and responsibility." He thinks elected school boards are fine if determined by a local referendum and given taxing power.

And with the state enjoying a triple A bond rating, the new sena-

tor can see no reason, he volunteered, to avoid floating bonds for education. "The students who use the facilities will get jobs, pay taxes and help pay off the bonds," he explained.

On current medical issues, he placed himself squarely against the

sale of body organs. If a bill on mastectomies could be made agreeable to both the medical community and the public, it could pass, he predicted. He expressed the belief that any proposals having to do with reimbursement by diagnosis-related groups, or DRGs, should be

## Component societies elect new leaders

VIRGINIA MEDICAL'S coming August issue will consist of a new edition of the Directory of Medical Society of Virginia members first published in 1982. Featured in that directory will be a listing of all the officers of Virginia's 46 component medical societies. Pending that inclusive index to medical leadership in Virginia, we present the names of these new presidents:

*Accomack County Medical Society:* **Dr. Parker C. Dooley**, Onley.

*Albemarle County Medical Society:* **Dr. William A. Orr**, Charlottesville.

*Alexandria Medical Society:* **Dr. Antonio M. Longo**, Alexandria.

*Alleghany-Bath Medical Society:* **Dr. Unity M. Powell**, Covington.

*Augusta County Medical Society:* **Dr. John P. Heatwole**, Waynesboro.

*Culpeper County Medical Society:* **Dr. George E. Broman, Jr.**, Culpeper.

*Fredericksburg Area Medical Society:* **Dr. Stewert E. Kohler**, Fredericksburg.

*Mid-Tidewater Medical Soci-*

*ety:* **Dr. Bruce M. Bucher**, Tappahannock.

*Newport News Medical Society:* **Dr. Donald H. Chessen**, Newport News.

*Northampton County Medical Society:* **Dr. Robert L. Erdman**, Nassawadox.

*Northern Neck Medical Society:* **Dr. Robert W. Poole**, Killmarnock.

*Northern Virginia Medical Society:* **Dr. Terry L. Sinclair**, Winchester.

*Prince William Medical Society:* **Dr. Kathleen Zaremba**, Woodbridge.

*Richmond Academy of Medicine:* **Dr. Wyatt S. Beazley III**, Richmond.

*Rockingham County Medical Society:* **Dr. Richard M. Senfield**, Harrisonburg.

*Southside Virginia Medical Society:* **Dr. Theodore C. Andrew**, Hopewell.

*Tri-County Medical Society:* **Dr. J. Mills Britt, Jr.**, Franklin.

*Virginia Beach Medical Society:* **Dr. Richard A. Mladick**, Virginia Beach.

Reelected to serve the *Bedford County Medical Society:* **Dr. John A. Wentz**, Bedford.

watched carefully. "The people who come up with these ideas do not understand the practice of medicine," he emphasized.

He went on record as hoping the legislature would take no action on uranium mining in Virginia without more study of all the issues involved. He thinks the state's mental health services need dramatic improvement. He would vote for upping the state sales tax by 1¢ if the increase could go directly to mental health, the elderly, or education.

Clancy Holland brings to the Virginia Senate a thoroughly Virginia background. Born in Isle of Wight, he was educated at Hampden-Sydney College and the Medical College of Virginia, Class of 1958. Even his tour of duty in the Navy as a line officer wound up in Virginia, at the Portsmouth shipyard. He married Mary Burton of Suffolk; they have three daughters, all now in their 20s.

Public office has taken a heavy toll of his time with his family, to Dr. Holland's regret. When he was mayor of Virginia Beach, the speech-making and meeting and greeting kept him running from hospital rounds to office patients to home for a change of tie before going out again at night. Now, as a state senator, he has encountered the constant barrage of functions given by lobbyists from breakfast to dinnertime.

Yet, he told his interviewer, he loves the constant activity and has learned to enjoy the spotlight.

"When I was mayor, I was riding on a parade float one day, all decked out as King Neptune for the Neptune Festival, and I was just thoroughly enjoying myself, when someone came up to me and asked me how in the world I could stand doing that."

He chuckled. "I guess it's the ham in me," he said.

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## Varied agenda occupies councilors in first 1984 session

The meeting of The Medical Society of Virginia's councilors on January 14 was President C. Barrie Cook's premier appearance as the Council's presiding officer, and he prefaced the agenda with an introduction. Emphasizing the key roles played in organized medicine by communication and participation (cf *Va Med* 1984;111:61), he stressed the importance of the work of the Society's committees. Then he revealed that he had asked the Long Range Planning Committee to review 1) the services the Society offers to its members and 2) the Society's interaction with the alternative delivery systems that are proliferating in Virginia.

The councilors proceeded to take these actions:

- Approved an appropriation of up to \$10,000 for a professional consultant to serve the Medical Society of Virginia Review Organization,

---

## Resident #1 signs in

The new Resident Physicians Section of The Medical Society of Virginia's House of Delegates has its first member. He is Sam Barton, MD, who is now in Blackstone, Virginia, performing his family practice residency. A 1982 graduate of the Medical College of Virginia, Dr. Barton was active in the student medical society and was a delegate to annual meetings of The Medical Society of Virginia.

All interns, residents and fellows at work in Virginia will be invited to join the new section in a mailing scheduled to go out early this month. The dues are \$10 per year, for which minimal sum those who join will have all the rights and privileges of active MSV members.

which is expected to begin functioning in October 1984. The consultant's initial assignment will be to develop the requisite proposal for a contract with the Department of Health and Human Services that will establish the MSVRO as the peer review body for Virginia.

- Went on record as opposing any change in the Medicaid program that would remove the ultimate responsibility for the program from the State Department of Health.

The Council heard more on Medicaid. Dr. Cook reported that legal support has been given by the Health Care Financing Administration's general counsel to the Medicaid program's position that a Medicaid recipient is not responsible for an indebtedness resulting from adverse medical necessity determinations for covered services; this relates to inpatient hospital care exceeding 21 days per admission, and services through the 21st day if they are not considered medically necessary.

Dr. Cook also noted reports of coming changes in the Medicaid program involving second opinions for elective surgery and ambulatory status for certain surgical procedures.

- Approved a recommendation from the Membership Committee that the Society create a Hospital Medical Staff Section within its House of Delegates. The councilors directed the Membership Committee to develop an implementation plan and the Bylaws Committee to prepare enabling amendments.

Also suggested by the Membership Committee was a change in the age at which dues-exempt status is conferred on retired members—from 70 years, as now in





An oil portrait of Robert I. Howard, former Executive Vice President of The Medical Society of Virginia, was unveiled at the meeting of Council in January. Dr. Thomas M. Wright, Fairfax surgeon and portraitist, had been commissioned to paint the likeness and was on hand when it was revealed by Dr. C. Barrie Cook, MSV President. Shown above are, from left, Dr. Wright, Mr. Howard and Dr. Cook. The genial Scotsman retired in December 1981 following 30 years as the Society's professional helmsman. The gilt-framed portrait will hang at MSV headquarters.

effect, to 65 years, with the provision that the physician has been a member for the ten preceding years. The councilors wanted to know how many MSV members are in the 65-to-70 age bracket, before making a decision on this proposal, and asked the committee to supply that data.

- Agreed on Thursday, May 17, as the date for the annual Congressional Luncheon in Washington and approved tentative plans for briefings by representatives of the Federal Emergency Management Administration and the Defense Department and a tour of the Pentagon.

- Endorsed the nomination of Dr. John P. McDade, Alexandria, for a seat on the AMA Council of Legislation.

Other matters brought to the Council's attention:

At the American Medical Association's meeting in June, Dr. William S. Hotchkiss will be seeking reelection to his third term on the AMA's Board of Trustees, of which he is

Vice Chairman. Dr. Hotchkiss was MSV President in 1972.

The councilors heard that at the Insurance Committee's request, the St. Paul Companies, carrier for The Medical Society of Virginia's professional liability insurance program, tentatively agreed to a realignment of the territories by which experience is rated. Under the change some subscribers' rates would go up and some would go down, with the net result of more equitable rates for all.

Affected by the plan are the five counties of Charles City, Dinwiddie, Hanover, New Kent and Prince George, and the three cities of Colonial Heights, Hopewell and Petersburg; they would be transferred out of Territory 2 (Norfolk) and into Territory 4 (Richmond). Also, Gloucester County and Poquoson city would move from Territory 3 to the Norfolk territory. The proposed revisions must be filed with the State Bureau of Insurance. They are expected to go into effect no later than July 1, 1984.

# The Medical Society of Virginia

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## From the President: Utilization and Quality Control

**H**ospital review committees and numerous governmental guidelines are nothing new. For some years we have had hospital committees of various types, i.e., infection control, tissue, transfusion, safety, etc. The impact and effectiveness of these committees has varied greatly.

In truth, many of these committees have had minimum impact on bed utilization as that was not their main purpose. Their main concern was quality of care. However, they have made medical staffs aware that someone was indeed looking at what and how they were performing their functions as practicing physicians. This has helped improve the quality of care patients received. The better these committees functioned, the better care many patients might expect to receive and the better the reputations these hospitals might develop. As a result, more physicians and patients wanted to utilize those hospitals.

Now DRGs (diagnosis related groups) are upon us as a result of the Tax Equalization and Fiscal Responsibility Act of 1982 (TEFRA) and the Social Security reform legislation of 1983. Hospitals now will be reimbursed for Medicare patients according to a predetermined schedule by diagnosis. The utilization review committee will be responsible for determining the allowable time in hospital and the expense incurred in relation to the diagnosis. They will encourage you to discharge the patient as fast as possible with as few x-rays and tests as are absolutely necessary because these areas are now cost centers rather than profit centers.

The problem that faces us now is to make sure the patient does not get short-changed, does not get poor quality of care, and is not in any way damaged by being rushed through the period of hospitalization. On the other side of the fence, we need to be concerned about the hospital. And we must try not to keep the patient longer than necessary, thus causing the hospital financial hardship. Such hardship could ultimately cause the hospital to be financially insolvent or unable to refurbish buildings, obtain new equipment, or secure a proficient, up-to-date nursing staff.

At the same time, those hospital committees currently charged with maintaining quality care will find their role more demanding and even more important than in the past.

Our challenge is to accomplish this for the benefit of all concerned.

**C. Barrie Cook, MD, President,  
The Medical Society of Virginia**

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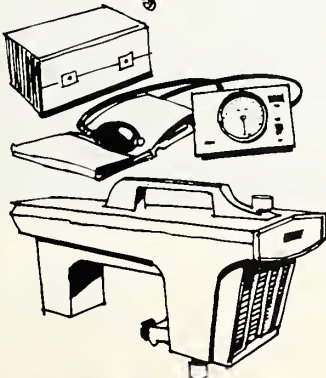
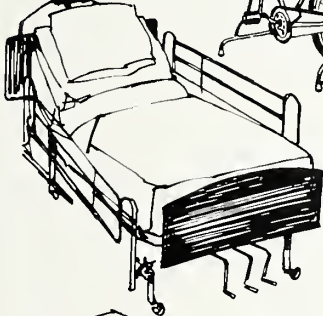
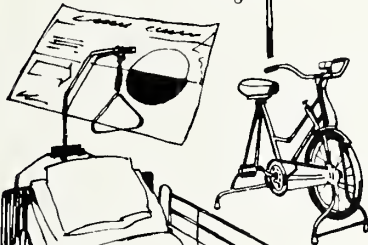
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# On legislature's eve, PAC gives a party

'Twas the night before the General Assembly's opening, and all through the Grand Ballroom of the Commonwealth Club in Richmond plenty of people were stirring at the second annual reception given for Virginia legislators by the Virginia Medical Political Action Committee's PAC 250 members. Lots of legislators showed up, to be greeted at the door by Dr. Harold L. Williams, VaMPAC chairman, and then proceed to the bar, the buffet table, and the physicians who awaited them.

Virginia's second in command, Lt. Gov. Richard J. Davis, who is also the President of the Senate, was there. So was the Speaker of the House, A. L. Philpott. And just as the party was winding down, in walked the Governor himself, Charles S. Robb, to circulate about the room and edge up to the buffet table for a bite to eat.

Here and on the next two pages are pictures of some of the PAC members and legislators who were there.

*Photographs by Tim Wright*



▲ Dr. Norman R. Edwards, Hampton (left), with Del. Arthur R. Giesen, Jr. (R-Verona). Between them is Lt. Gov. Richard J. Davis.



▲ Dr. Robert M. Kesler, Norfolk (right), and Sen. Stanley C. Walker (D-Norfolk). At rear, Del. Mary A. Marshall (D-Arlington).



▲ Dr. Custis L. Coleman, Richmond (left), with the Attorney General, Gerald Baliles. At rear, veteran Sen. Edward E. Willey.





The new senator  
from Virginia Beach,  
Dr. Clarence A. Holland (left),  
with Dr. Iver Kasenetz,  
Falls Church.  
For more on Senator Holland,  
see page 118.

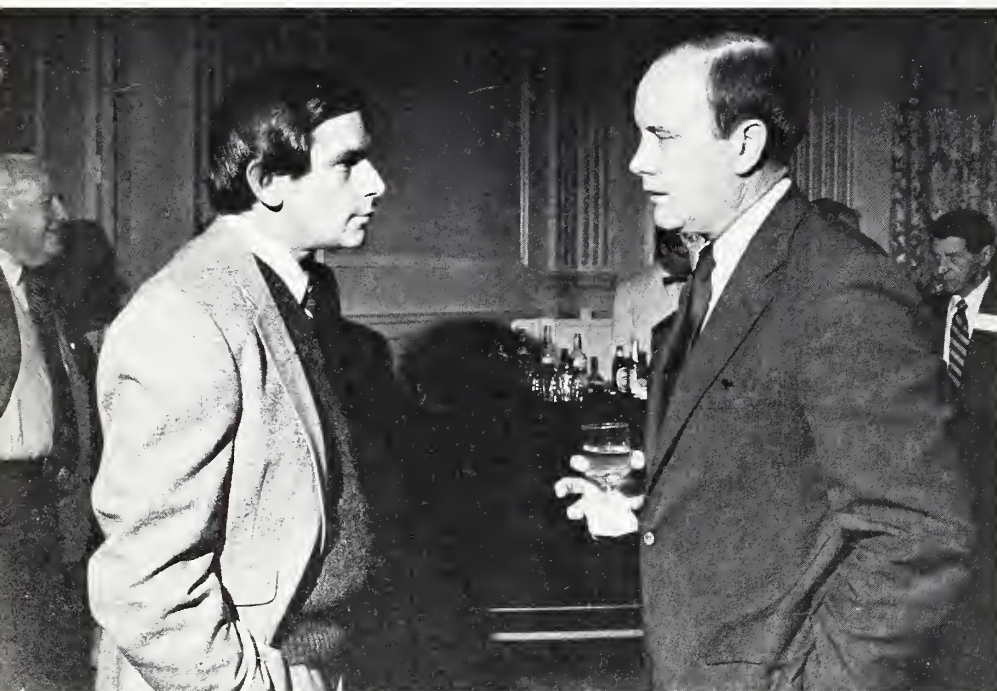




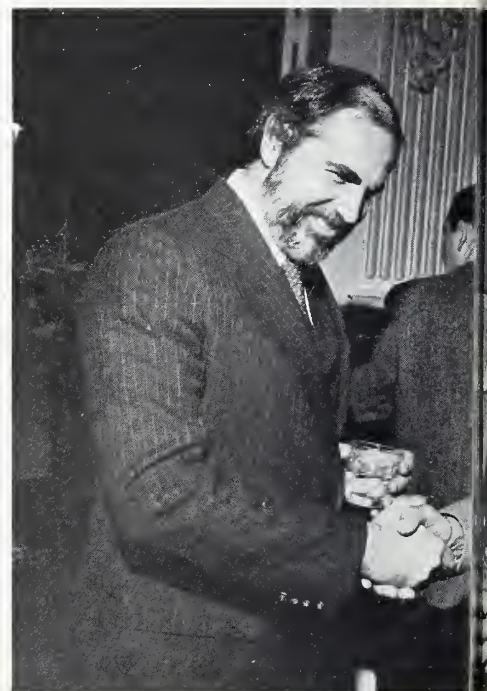
▲ Dr. Antonio M. Longo, Alexandria (right), with Speaker of the House A. L. Philpott (D-Bassett).



Dr. Lindley T. Smith, Richmond (left), with Lt. Gov. Richard J. Davis



▲ Dr. John R. Partridge, Richmond (left), and Del. Owen B. Pickett (D-Virginia Beach).



▲ Dr. Salvatore D. Barranco, Blacksburg (left), brother of freshman Sen. Clarence





▲ Dr. Richard E. Palmer, Alexandria (right), and Del. C. Hardaway Marks, (D-Hopewell).



▲ Dr. Gerald C. Burnett, South Boston (left) with Del. Vance Wilkins (R-Amherst).

ets Sen. Richard J. Holland (D-Isle of  
red



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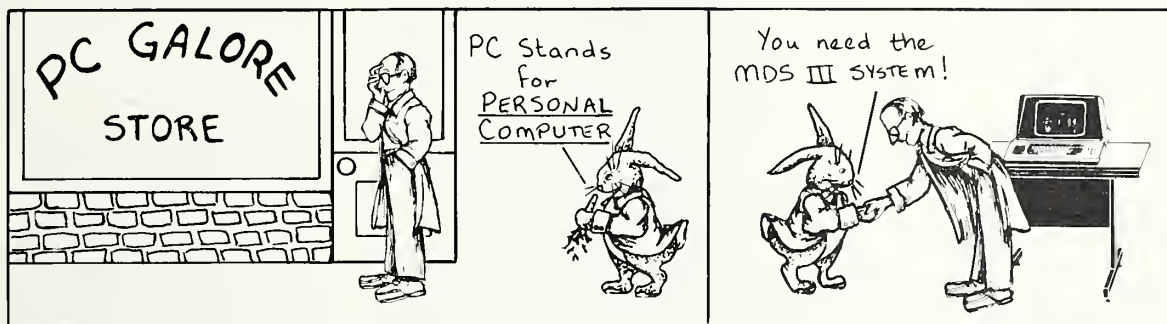


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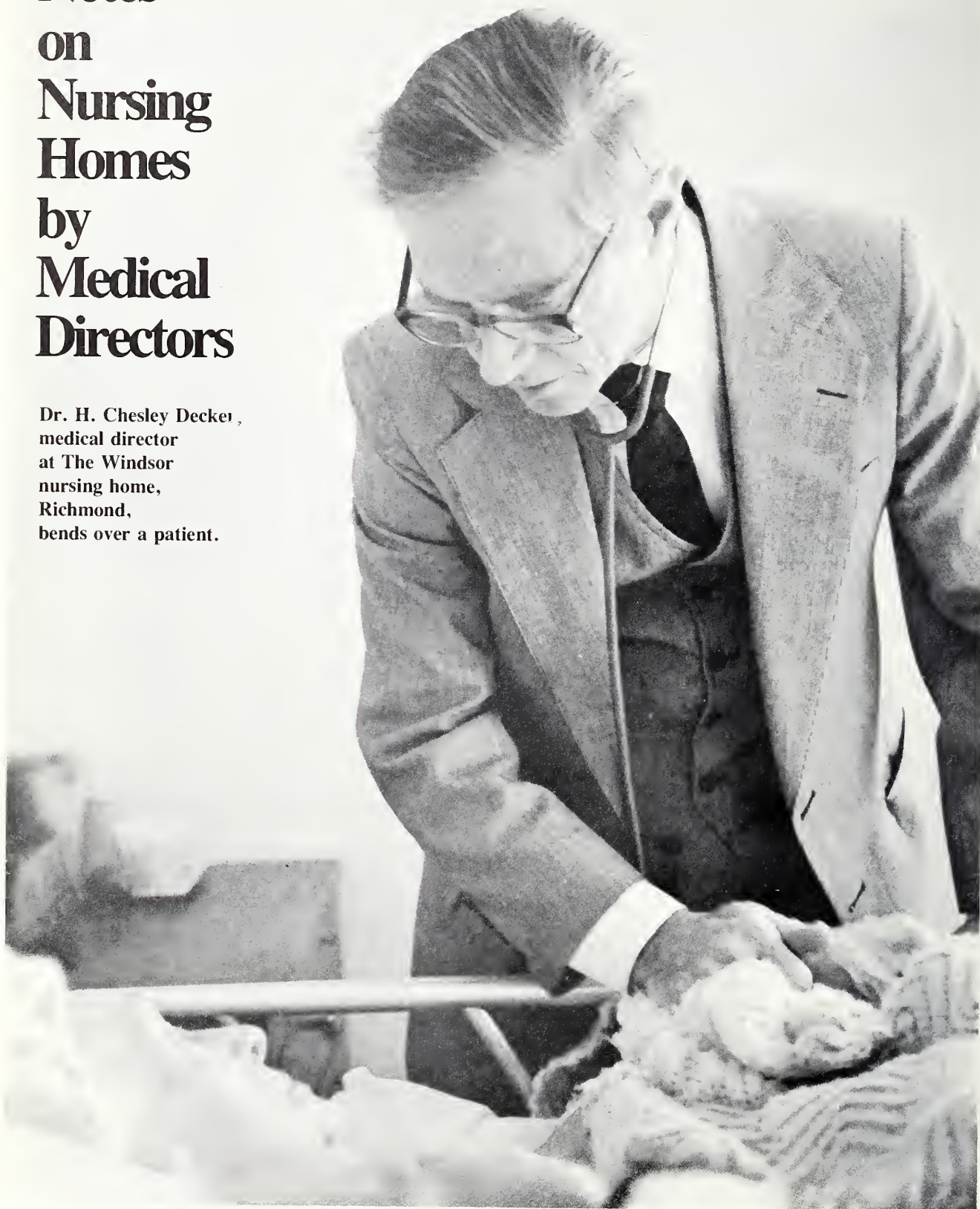


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# Notes on Nursing Homes by Medical Directors

Dr. H. Chesley Decker,  
medical director  
at The Windsor  
nursing home,  
Richmond,  
bends over a patient.





AT HIS DESK in the Virginia Department of Health, Dr. Robert L. Wood was thinking about Virginia's nursing homes. The facilities themselves were not what he was brooding about; from the reports of health department inspectors Dr. Wood knew that compliance with rules and regulations was good, and there had not been, of late, one of those unfortunate accidents that the media loves to spin into sob stories.

By federal law enacted in 1972, every private nursing home must designate a licensed physician as medical director, and it was these medical directors that concerned Dr. Wood, who was Medical Program Director of the Office of Health Planning and Resources Development.

With no regular pipeline between these physicians and the Department of Health, Dr. Wood had no answers for the questions going through his mind. What were these medical directors doing and thinking? What were their problems and beefs? Was there something that could be done to help them?

Dr. Wood set about devising a questionnaire to get some answers.

A physician may serve more than one nursing home as medical director. Thus the record then showed 179 nursing homes and 158 medical directors in Virginia. Dr. Wood mailed his questionnaire to each physician. He received in return 109 completed forms, a response rate of 69%.

OF THE MEDICAL DIRECTORS who responded, 25 said they direct the medical care of relatively small facilities with 60 beds or less. Six said they serve large institutions of 240 beds or more. The sizes of the other respondents' facilities ranged in between.

How are these directors paid? The questionnaires revealed that 60 of the respondents have annual

## VIRGINIA MEDICAL

MARCH 1984

contracts with the nursing homes they serve, and 19 are paid by honoraria. Of these 79 paid directors, ten said they serve more than one facility and so collect more than one check. Of the remaining respondents, 32, or 29%, said they had been chosen by medical staffs and received no pay at all.

What are the hours of work? "One to eight hours per month," reported 62 medical directors. In sharp contrast, "41 to 60 hours," was the estimate given by six. Of those with hours somewhere in between, 31 reported they work nine to 24 hours monthly and seven estimated they put in 24 to 40 hours of medical directing per month.

Do they admit patients? Yes, said 93 of the respondents. No, said 16.

The questionnaire offered a list of duties commonly associated with the medical direction of a nursing home and asked the physicians to check those they performed. All but five of the 109 respondents checked "advise on development and execution of patient-care policies." The other duties most frequently checked were:

- provide consultation on infection control and isolation procedures;
- provide temporary or emergency medical services;
- advise on the suitability for admission of patients;
- advise on the employees' health program;
- act as liaison between the nursing home's administrator and attending physicians.

"What are your concerns?" Dr. Wood's questionnaire then asked. The answering doctors let loose with mingled praise and pain.

THE PRAISE, when it came, was comprehensive.

"An excellent facility with no major problems," was the way one medical director summed up his charge. Another described his facility as "running very smoothly", while another observed, "Our small, rural nursing home is so well run by the administrator and the board that I have had very few problems."

One medical director credited his problem-free institution to the "stable nature of the administration and staff." Another thought "cordial professional relations" were responsible for the lack of problems during his ten months on the job.

"Patients are receiving good nursing care, and meals are very satisfactory. Fortunately, problems are few," another respondent reported. Yet another medical director described himself in the happy position of having "no concerns. It is an excellent facility with no major problems."

One medical director reserved his praise for Dr. Wood's survey. After citing several problems of substance he had experienced in his nursing home, he congratulated Dr. Wood for his interest because "in my opinion these areas need to be looked into."

OF THE CRIES of pain registered by respondents to the survey, the largest and most anguished chorus sang out "PAPERWORK", with the rigors of state and federal regulations providing variations on that theme. Because so much of their care is paid for with public funds, nursing homes are watchdogged relentlessly. Among the myriad paper processes, the recertification of patients is a particular plague, but there are endless other forms to fill out as well. For instance, patient visitation is now mandated by law, every 30 days for





A nurse accompanies Dr. Donald S. Thorn, medical director, Sleepy Hollow Manor Nursing Home, Annandale, as he looks in on a patient.

Medicare patients, every 60 days for Medicaid patients, every 90 days for private-pay patients, whether the visit is medically indicated or not.

As one respondent summed up this chief complaint: "Too much attention to forms and reports, too little to patient care." Another called Medicare and Medicaid documentation "busywork for the inspectors," and another referred to the "ever-encumbering" directives from federal and state governments.

"We need," said one medical director, "to formulate uniform approaches of coping with this huge, unnecessary paperwork."

One medical director sympathized with his attending physicians' reluctance to put pen to paper: "The major problem is compliance with unnecessary state and federal rules, which require physicians to come in and do bureaucratically generated paperwork. They will not do this, and I cannot blame them, but from the nursing home's viewpoint, it puts those in charge of trying to follow these absurd requirements in a no-win situation."

As for the visitation regulations themselves, these are "arbitrary, useless, and expensive requirements that patients be seen whether needed or not," said one survey respondent.

"Wasteful of taxpayer money and physician time," observed another medical director and added, "We have good nurses and can rely on them to call the physician when necessary."

Still another respondent was convinced that "these regulations, as interpreted and applied by various inspectors, tend to address the performance of mandated activities rather than an assessment of patient care outcome."

And one doctor lost his temper, denouncing the "idiotic, picayune



Dr. George Stanley Mitchell, Jr., medical director at the James River Convalescent Center, Newport News, ponders a patient's complaint.



requirements and directives that spew out of ivory towers . . . to the end result that caring for the patients is rendered nearly impossible."

**T**HE QUALITY of care delivered in their nursing homes deeply concerned many of the medical directors who replied to Dr. Wood's questionnaire.

"The comfort and well-being of the patients," is the way one respondent described it. Another referred to it as "the general contentment of all patients," who in his facility, he noted, averaged over 85 years of age.

A lot of frustration was expressed in this area. "I do not have sufficient time to do as much as could be accomplished in this position," one respondent confided. Another said he acts as attending physician for 90% of his facility's patients. Another "takes care of the sick and makes rounds on about 55 patients at least once every two months, admitting patients to hospital as needed."

But other medical directors don't have the chance to do much. "Under the present setup," said one survey respondent, "I have no real authority and little day-to-day contact to see how well the patients are cared for. We have regular meetings . . . at which I inquire and advise, which is really all I can do."

Another respondent had much the same problem. In his proprietary facility, he said, "management rarely seeks substantive medical direction from me. I'm more of a clean-up, trouble-shooter sort of help."

One medical director felt the need to "make sure that patients receive adequate care as economically as possible," reflecting the tight rein on costs in nursing homes today. Another was worried because "our 58-bed nursing home is 25 or more years old and in many



**Dr. Anthony R. Stavola, medical director, talks over a problem with a patient at the Friendship Manor Convalescent Center in Roanoke.**

ways does not meet present-day standards."

Asserting that "nursing home administrators resist turnover of 'desirable', or low-care, patients to lesser care," another medical director saw a need to establish "realistic goals, so that intermediate care does not become a dead-end street with no goal but maintenance."

**T**HE RANGE of other concerns expressed by the medical directors who answered Dr. Wood's survey reflects the many factors affecting their responsibilities.

Admissions is a thorny topic, with several physicians echoing one medical director's complaint that he has "no authority regarding the suitability of patients to be admitted."

In that connection, "hard-to-handle" alcoholics were listed as a problem for one medical director because, he wrote, "patients are transferred from other institutions without contacting me regarding diagnosis, Rx, needs, physical therapy, etc."

Another was worried about admitting to an extended care facility "mentally ill patients and those needing total care because of men-

tal incapacity. The atmosphere for the other residents could become very unpleasant if not threatening," he asserted.

Shortages of registered nurses and other support figures bothered many of the respondents, and only one of the medical directors painted a bright personnel picture: "Intelligent, industrious, and kind-

hearted employees are the key to success in proper treatment of chronic diseases, especially in the elderly," he observed, "and I feel we have that at this facility."

Others lamented the low morale and high turnover of nursing home employees. "Many of our employees are overworked, underpaid, and not appreciated," said one.

Whether interested in their elderly relatives or not, the families of nursing home patients appear to pose problems. One director regretted that families extend so little interest, but according to another respondent, when they are interested, their "expectations are often too high," and another noted that families "expect daily visits by

## Virginia's old folks at home

**I**N STEP with a national trend that has made nursing homes "the centerpiece of the long-term-care system,"<sup>1</sup> Virginia's nursing homes can now accommodate almost twice as many people as they could ten years ago. The figures: In 1973 there were 167 homes with a total of 10,948 beds. In 1983 there were 183 homes with 20,020 beds.<sup>2</sup> At last count the beds were 98% occupied.<sup>2</sup> The occupants were almost entirely the elderly, with an occasional sick or handicapped younger person.

The State of Virginia defines a nursing home as any facility, whether free-standing or not, that is licensed by the Virginia Department of Health to provide 24-hour supervision, personal assistance and nursing care. Eleven of the acute-care hospitals in the state have nursing-home units.

Most of Virginia's nursing homes are separate entities, however, and these are of two kinds: 1) proprietary enterprises operated for profit, and 2) non-profit homes run by religious and fraternal organizations. It is the for-profit nursing homes that have shown such dramatic recent growth, not in the number of homes themselves but in the number of people they can accommodate, or bed size.

Gone, now, are the small facilities of the "mom and pop" variety that once characterized Virginia's proprietary nursing homes. The big hospital chains started buying them up in 1972, when Medicaid reimbursement became available for long-term care, and expanded their acquisitions with new beds, lots of them. Thanks largely to the power of modern American medicine, the supply of elderly survivors is everywhere on the increase, so occupants for the new beds were not hard to find. At the same time, the cost of medical care was bounding upward.

As a result, it's a lot easier to get into a nursing home in Virginia these days, but it's a lot more

expensive to stay there. In fact, Virginia's nursing homes are now big business, with gross revenues for the fiscal year ending July 1983 estimated at \$256 million.<sup>3</sup>

Of that total, Medicaid paid \$174 million. That was more than double the 1977-1978 Medicaid payout of \$83 million.<sup>3</sup> The taxpayers furnished the Medicaid funds, through the federal government (three-quarters) and the state (one-quarter).

Some of what Medicaid didn't pay for was picked up by Medicare, another taxpayer source, but most of it came from private payers. These were not only the patients themselves or their families, but also the Baptist and Methodist churches, which operate 1,650 nursing home beds in Virginia and refuse public funding, partly because of their interpretation of the separation of church and state and partly because of their conviction that they should look after their own.

**W**HAT do nursing homes charge per patient these days? That depends on the relative luxury or simplicity of the home and on what kind of care the doctor orders.

Cost of Care in Virginia's 121 Proprietary Nursing Homes,<sup>2,4</sup>

	One Day		30 Days	
	Range	Average	Range	Average
Skilled Care	\$40-\$89	\$49	\$1200-\$2670	\$1470
Intermediate Care	\$30-\$67	\$43	\$900-\$2010	\$1290

Skilled care comes closest to hospital care, with 24-hour nursing service. Convalescent patients and those with long-term illnesses usually need skilled care. Under certain conditions, Medicare will pick up the first 100 days of skilled care; then, if the patient is eligible, Medicaid funding may become operative.



MDs" and "demand frequent nursing attendance."

Of nursing home food, it's "unappetizing," tersely opined one medical director; moreover, he added, "blind patients and those deformed severely by arthritis often have to wait to be fed until the food is cold."

Another director asked, "Should

the level of nutrition be assessed solely by periodic weight measurement and the physician's visit every 60 days, or should there be other criteria, such as calorie count?"

Clinical procedures were questioned by many of the responding medical directors. One deplored the lack of standards for tube feed-

ings. Another expressed the view that the administration of intravenous medication and fluids should be permitted in his intermediate facility, where there are RNs and LPNs around the clock, "instead of calling an ambulance and sending the patient to a hospital for such treatment."

The need at his facility for

Intermediate care signifies less extensive services for those persons who are not capable of living by themselves but not sick enough to need 24-hour nursing care. Only Medicaid covers intermediate care. The overwhelming majority of nursing home beds in Virginia, over 90%, are certified for intermediate care.

For their charges, Virginia's nursing homes must by law provide 1) bed and board, 2) nursing care supervised by a registered nurse. Some frequently needed services that may be included in the daily rate or may be charged as extras are personal laundry, medical equipment, nursing supplies, physical therapy, transportation.<sup>2</sup> Medicines and the fees of private nurses are always extras.

The law also requires that the home must employ a licensed physician as medical director to keep watch on the quality of care given the patients. And every resident of a nursing home must have a personal physician who visits on a regular basis and certifies to Medicaid or Medicare the patient's need for the nursing home environment.

Myriad other federal and state regulations, including a Patients' Bill of Rights,<sup>5</sup> discipline the conduct of Virginia's nursing homes. The state monitors compliance, through masses of paperwork and inspectors from the Department of Health.

**T**HERE are now 21,560 persons living in Virginia's nursing homes.<sup>2</sup> The great majority are over 65; the median age is 82. The average length of stay is 1½ years.<sup>3</sup>

Not only are the bodies of these old folks wearing out after long use, but they are also assailed by chronic diseases.

Chief among these diseases are heart and circulatory problems, including strokes; arthritis; and cognitive disorders, which include the deterioration of memory and function diagnosed as senile dementia or Alzheimer's disease, and multi-infarct dementia, which results from a series of strokes. Some profes-

sionals consider Alzheimer's disease to be the single most common condition putting old people into American nursing homes.<sup>6</sup> A booklet designed to help families cope with the Alzheimer's disease patient has become a best seller, as see page 156 of this issue.

And these brochures can be recommended by the physician when patients or their families want to know more about nursing homes:<sup>7</sup>

- "Thinking About a Nursing Home." Free from the American Health Care Association, 1200 Fifteenth Street NW, Washington DC 20005.

- "Selecting a Nursing Home." Free from Concerned Relatives of Nursing Home Patients, PO Box 18820, Cleveland Heights OH 44118.

- "Tips on Choosing a Long-Term Facility." Send 25 cents and a stamped, self-addressed, business-size envelope to the Council of Better Business Bureaus, 1515 Wilson Boulevard, Arlington VA 22209.

—ANN GRAY

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the available facilities, such as recreation, rehabilitation, physiotherapy?"

A colleague was disturbed by the costliness of the system. It's a "tremendous burden, supporting Medicaid patients in these expensive beds," he stated. "The taxpayers cannot afford it."

A third respondent was gravely concerned about "the difficulty of providing care for patients who are in the end stages of their degenerative diseases, where, truly, there is no hope of salvage to any useful purpose."

"A time comes when one should be allowed to die with dignity, but guilt on the part of the families in institutionalizing their next of kin interferes with proper judgment in discussing, and allowing, their loved ones to die gracefully."

"I believe," this respondent concluded, "that many of our efforts serve little purpose other than to increase the expense to all."

Similarly, another medical director registered these sentiments: "I wish more doctors would discuss with relatives of the terminally ill patient, or the patient with advanced brain damage, the use of simple medications for comfort care only." In fact, he added, he is in favor of a general reduction of the intensity of care for these patients.

And the experience of one medical director had apparently been so negative that he was moved to this commentary: "The nursing home as it presently exists is, in my opinion, a tragedy and a reproach to our society. In large measure this is because of the widespread use of these facilities as dumping grounds for persons who could be much better cared for elsewhere."

—ANN GRAY

Dr. Wood is now Director of Medical Support of the Medicaid Program, State Department of Health.



**Dr. William J. Hagood, Jr., medical director at The Woodview nursing home in South Boston, visits a patient.**

"swing beds" between skilled and intermediate care concerned one respondent, while another asked for "much more specific definitions" of the categories of care.

"These are days when patients are sent home with self-administered hyperalimentation, decubitus care, etc., under the guidance of the family," he observed, "and there is no reason why intermediate care facilities cannot undertake cases involving ostomy care, enteral and parenteral hyperalimentations, etc."

Some of the responding directors registered complaints about their peers, the attending physicians, and usually related the difficulties to "getting physicians to sign or-

ders and complete charts at the proper times" but with attendance at medical advisory sessions also a sore subject.

"Training and workshops in geriatric care," was the felt need of one medical director, while another thought meetings with Virginia's other medical directors might be helpful.

**L**ARGER ISSUES were addressed by some of the medical directors who responded to Dr. Wood's survey.

Asked one medical director, who described himself as the only physician caring for the patients at an intermediate care facility: "Is the quality of life being promoted with



# Nursing home care can be fun

I WAS DELIGHTED with a recent *New England Journal of Medicine* "Sounding Board" piece<sup>1</sup> in which the position was taken that nursing homes would improve in quality of care if only good practicing physicians were put in charge. My pleasure in Ted Schwartz's paper undoubtedly stems from the fact that I've served for three years now as part-time medical director of a top-notch long-term care facility, one in which 135 female residents . . . constantly test our mettle.

The home where I work is fortunate in not having many of the usual problems of nursing homes; it has a dedicated board of trustees, ample—if diminishing—resources, a roster of devoted medical consultants, a sound administration, a superb nursing staff, and a complement of cheerful and competent technicians and employees. It follows that my job, working in coordination with another practicing internist, is a straightforward one: to provide the residents with quality care.

I would like to share some impressions I've gained from my work at the home:

1. Old people have just as many acute ailments (colds, diarrheas, rashes, sprains) as young people; managing these is not different, except for the need to be careful about dosage of medicines.

2. Old people often have several overlapping chronic diseases, the combined management of which is enough to tax the technical skills of any internist.

3. Old people usually have a realistic understanding about disease and its management, but what they really care about is function—seeing, hearing, eating, ambulating, conversing, staying continent and in general being well enough to participate in the array of organized activities available to them.

4. Any change—in body function or behavior—may signal new disease and warrant investigation. If the nurse says, "Something is going on with Mrs. Blank," it behooves the physician to pay attention.

5. But when senile dementia dominates the picture, it is unnecessary—providing family members and nursing staff concur—to pursue thoroughly every new event.

6. Senile dementia itself, although admittedly a

special problem, can be managed, partly with judicious use of drugs, but mostly through constant supervision by caring nurses.

7. Old people often have a stubborn hold on life and are capable of withstanding an astonishing amount of stress. At times, the hold is tenuous and life can be snuffed out by a seemingly trivial occurrence.

8. It is usually better to die in the nursing home than at the hospital.

9. Team care helps old people. It is the physician, therapists, dieticians, aides, lab technicians and most of all, the nurses who make the medical care work.

10. We need useful clinical research about osteoporosis and its fearsome sequelae, and about vertigo and inner ear disturbances (we call them "the dizzies"). We need scientific confirmation that it is OK to use shotgun antibiotics for mysterious fevers, and to leave asymptomatic urinary tract infections untreated.

11. Risk factors may not be as important to longevity as is proclaimed. Our universe in the home contains smokers, tipplers, the overweight, the sedentary, and Type A personalities, not to mention those with diabetes, hypertension, previous cancers, high cholesterol and sluggish bowels. A high proportion of residents report they have lived longer than any of their relatives. The truth is we're dealing with survivors.

12. With rare exceptions, clinical judgment is better than lab tests.

13. Old people seem to have a better sense of humor—or display it more freely—than the rest of us.

14. Taking care of old people in a nursing home is intellectually interesting and emotionally satisfying.

15. It is also fun.

WILLIAM CAMPBELL FELCH, MD

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# Adult Day-Care Centers in Virginia

John R. Taylor, MD, *Richmond, Virginia*

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A select group of aged persons can benefit from the services of adult day-care centers, where, the author observes, association with their peers and group activities often gives them a new enthusiasm for life, while their caretakers welcome the daytime relief.

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**W**HEN PRIMATES achieved the biologic uniqueness of regularly surviving beyond their years of vitality and reproductive capability, and man has succeeded in this better than any other animal, an entirely different set of problems accrued. For the human civilization, these issues are social, and economic, and include mental and physical health. The extent of resolution of these issues, either by individuals or by society, determines the extent of family disruption, loss of aggregate family wealth, required institutionalization and other dislocations, poverty, starvation, and unnecessary ill health that ensue.

Given that the object of any comprehensive plan to address these problems will be retention of the integrity of the collective family unit, a number of remedies have evolved. One of them, the adult day-care center, is addressed here. By definition, these are centers housed in small to medium structures—

private homes, community centers, church education buildings, etc.—that provide, through a paid staff, care of infirm persons during a 9- or 10-hour daily period that brackets normal work hours. Weekends and holidays find such centers closed.

Credit for the development of the concept of the day hospital, at least in the modern era, is usually given to D. Ewen Cameron,<sup>1</sup> a psychiatrist who founded the Allen Memorial Institute in Montreal in 1946. Dr. Cameron's concept differed from the present day centers in that he sought to treat psychiatric patients in the early phase of their illness in order to avert admission to a traditional hospital with beds. The idea spread, and by 1949 Robert Menninger opened his Menninger Clinic Day Hospital in Topeka. Eventually, the movement enveloped neurologic illnesses as well, particularly as the deinstitutionization of such patients surged through the 1960s and 1970s; but as it did so, the physiognomy of the centers changed from a unit oriented to acute crisis designed to prevent acute hospitalization to one that included the concept of care of chronic patients to prevent nursing home placement.

From the Department of Neurology, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Taylor at Box 599, MCV Station, Richmond VA 23298.



Virginia presently has 14 such day-care centers, largely clustered in or near metropolitan areas. Each serves from 15 to 50 clients. Sponsorship of the centers ranges from private operations to churches, hospitals, and community/civic organizations. While some scholarships are available for indigent patients from federal sources such as funds provided by the Older Americans Act, from the community, and from private donations, the majority require a fee or partial fee from the client. This fee varies from \$7 to over \$20, including lunch; occasionally, a center is able to waive most of the fee. Medicare, Blue Cross and other traditional third-party carriers will not defray this cost. In Virginia, 76% of the revenue for the centers derives from fees from the client, federal and community sources, private donations, and volunteer agencies, such as United Way. Contributions, such as free rent, volunteer personnel, transportation, etc., provide an estimated value that accounts for 24% of operating resources.<sup>2</sup>

Adult day-care centers are licensed by the Virginia Department of Social Services. Licensing is based on such criteria as the physical size and nature of the building, including safety features, and the characteristics of the staff. The director usually has a background in occupational and recreational therapy, in social work, in counseling, or in nursing. Though not required to do so, all of the centers offer some services in addition to supervision. Many employ at least part-time physical and occupational therapists. One day center with which the author is familiar, Stuart Circle Center in Richmond, a center partially underwritten by several nearby churches, is managed by an occupational therapist and employs a nurse part time. Nearly all centers have a nurse available to dispense medications, take blood pressures, etc. Some are able to conscript talented volunteers to lead singing and other musical activities as well as to assist in sightseeing and shopping tours in the adjacent area. Other services include restorative services and family support.

The patients benefiting from the services of a day-care center derive from varied social and health backgrounds. If aged, as most are, their spouses are also infirm or have predeceased them, leaving them without adequate home care. In younger patients, aged 50 to 65, spouses are living but place the infirm member in a center while they work. The majority, however, have spouses who have predeceased them, and they are now living with children who work or who find respite in having the infirm person out of the home for several days weekly.

Economically, the background of most clients is

low and middle income. Wealthy persons seldom utilize the services of a center, though in some cases they would benefit from this diversion.

Medically, the majority of persons found in day-care centers suffer from some form of neurologic disease that has produced their dependency, though numerous other disorders coexist. Others are patients with heart disease, diabetic and dialysis patients (who require a structured environment to follow their diet), patients with chronic respiratory problems and patients with chronic psychosis.

The two most common diseases, though, are neurovascular disease and dementia of the Alzheimer's type (DAT). Though both neurologic, these two disorders present quite different problems for the day-care centers. The neurovascular patients, usually hemiparetic, have problems in mobility, but if they are able to sit, the centers usually can cope with them, given adequate personnel to assist with transfers and with feeding. Since the client is present only during the day, dressing is not a problem except with regards to toileting, and center staffs can ordinarily manage this. Alzheimer's patients, and some neurovascular patients, have another problem, abnormal behaviour, that is less readily overcome. Wandering can be prevented by locked doors or vigilant staff, and disorientation, constant ruminations about the past, and quiet delusory states are well tolerated.

The very heart of the center's social structure, however, is "togetherness," i.e., most activities are performed as a single large group or in two or three smaller groups, and it is this structure that requires exclusion of certain persons from the centers. Patients with Alzheimer's disease and other illnesses that alter mental status may be loud and vituperative, even bellicose, and are unacceptable to the centers unless the behaviour can be modified with psychoactive drugs.

Another complication common to this group is that of bladder incontinence. Patients chronically incontinent and wearing a catheter and leg or wheelchair bag present no problem to the centers; those not requiring a catheter, though periodically urinating with impropriety, much as a child, are unacceptable. This is a particularly common problem, since the simple solution of installing an indwelling catheter is both unacceptable on medical grounds, because of infection, and impractical, because the patient will pull it out.

The final group of patients unacceptable to the centers are those unable to sit up; bed patients have no place in day centers as presently constituted.

*continued over*

## Benefits

Though situations vary, most infirm oldsters who conform to the above criteria benefit from spending several days weekly in a different environment. These benefits include a break for the client, professional care, such as physical therapy, and improvement of mental confusion and depression. A benefit difficult to quantitate but no doubt very real in many cases is the improved nutrition provided by one well-balanced meal daily. Overall, nearly 70% of clients appear to benefit.<sup>3,4</sup> The close association with persons their own age and with similar backgrounds, as well as the group activities and trips into the community, seem to inject a new enthusiasm for life.

Benefits to the caregiver are noted in an even higher proportion—77% in one study.<sup>3</sup> The chief benefit is a break for the relative, creating an improved mental outlook and more "private" time. Other positive changes for the caregiver are reduced worry about the well-being of the client, ability to find employment, more time with his, or more often her, own family, and reduced family stress.<sup>5</sup>

## Problems

Given the benefits discussed, why does Virginia have only 14 day-care centers, serving only 400 to 500 clients, who represent less than 1% of the estimated 80,000 impaired older Virginians?<sup>5</sup> Such factors as ignorance of the existence of these services or initial reluctance of the client to participate come to mind, but the principal reason is cost.<sup>5</sup> Since at least a portion of the cost of operating the centers must be borne by the client, a daily fee, offsetting usual home operating expenses only by the provision of a single meal, exceeds the resources of many families. Even though 59% of clients meet the criteria for admission to intermediate care nursing homes, Medicare and other third-party carriers have been slow to embrace this less costly alternative.<sup>6</sup> To be sure, such carriers cannot engage in the support of domiciliary expenses, but more support for selected cases would be appropriate. Day-care centers are, in fact, cost effective when compared to other forms of institutional care, particularly state psychiatric hospitals.<sup>7,8</sup>

Other problems deter the use of day centers.<sup>5</sup> The physical and psychologic effort involved in dressing and getting the client ready to go to the center each day can be trying, especially to the caregiver. Transportation itself may be difficult or expensive. Since the centers are not economically feasible for less than 15 clients, small communities are so far removed from centers that transportation cost preclude their use. Finally, once transported, it is unrealistic for the client not to stay in the center most of the day, and some of the infirm find this duration physically and emotionally exhausting.

## Summary

Adult day-care centers offer services for a select group of older persons, who usually are infirm because of neurologic and related illnesses. The benefits include enhanced well-being, skilled care, and respite for the caregiver. That these centers are underutilized in Virginia is due to several complex but not insurmountable problems.

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# Rx: Rehabilitation

Charles H. Bonner, MD, *Richmond, Virginia*

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Therapy at a rehabilitation hospital can ease the elderly person's transition from an acute-care setting to the home and in the process repair the patient's self-confidence and hope. The author describes some clinical goals keyed to restoring function and mobility in the aged patient.

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**G**RANDMA and grandpa have no less need to be independent and useful simply because they're growing old. True, they are more likely to have heart disease, a stroke, an infection, a fracture or a drug interaction. So when they and the millions like them become ill, they could use a dose of rehabilitation as well as emergency and acute care.

Grandma and grandpa are hardly alone, as one per nine Americans is now 65 years old or older. This high percentage of elderly citizens should come as no surprise since, back in 1947, Dr. Howard Rusk, the father of rehabilitation medicine, made a remarkably close projection as part of his look ahead at the need to treat the growing numbers of the elderly.

Together with a longer life span, the elderly need a feeling of self-importance and hope. Changes in the health care delivery system will bring reduced utilization of hospitals and increased availability of outpatient and some health services. More emphasis is already being placed on helping the elderly

stay at home instead of being placed in an institution. I am certain the concept of home care is a great hope to elderly persons. People separated from their loved ones and their most comfortable surroundings often lose the will to live, for, after all, what does one really have to live for?

More people are returning home after some medical illness has decreased their physical capabilities, and they need to become better prepared. In short, their physicians must pay more attention to their needs for rehabilitation, and increasing numbers of physicians are.

For many patients, rehabilitation to help maximize function and teach self-care skills can begin shortly after the medical condition stabilizes. A key goal at this point centers on preparing a program so the patient can avoid nursing-home care. A rehabilitation hospital like Sheltering Arms can become a bridge on which patients pass as they travel from the acute hospital setting to their home. It's a step that intends to make unnecessary the detour to the nursing home that too often becomes a dead end.

The path, of course, certainly isn't primrose. Many obstacles, if unaddressed, could stand in the way of the elderly patient's return home, like problems with mentation, mobility, architectural barriers

Dr. Bonner is medical director of Sheltering Arms Rehabilitation Hospital, 1311 Palmyra Avenue, Richmond VA 23227. Address correspondence to him there.



Cyane B. Lowden

**Dr. Charles H. Bonner with a patient at Sheltering Arms Rehabilitation Hospital.**

ers, incontinence in bowel and bladder, and others. Moreover, will he or she fall out of bed and fracture a hip, forget to lock the wheelchair before transferring to the commode, leave a burner on and start a fire—in other words, be left alone safely?

With a proper rehabilitation program, elderly people are able to learn balance, to build strength and endurance, and to become independent in dressing, feeding and personal care. They can learn new techniques to adapt for lost bodily functions. Like their younger colleagues, they too can adapt.

They adapt with help from a medically supervised team of therapists.

- The physical therapist helps the elderly patient regain strength and endurance through administering the medically prescribed program of exercises. The physical therapist might use ultrasound, electrotherapy or other physical modalities as prescribed to treat various medical conditions. The physical therapist would also help the patient learn to walk with a cane, walker or crutches and, if necessary, to use a brace or prosthesis. If walking is not possible, the therapist would teach both patient

and family how to manage a wheelchair safely and efficiently.

- The occupational therapist works to convert the patient's regained muscle tone into the capability to perform the functional activities of daily living. It is easy to identify quickly the goal of a physical therapist in exercising a specific muscle, but it isn't quite that clear to see the purpose of each of the occupational therapist's assignments. When a patient makes a belt or taps a copper bowl or stacks cardboard shaped to resemble ice cream cones, the reason why might seem vague. But each task is correlated within the design of instilling not only the capability but also the confidence to proceed without assistance.

If eating and dressing skills must be relearned, the occupational therapist might call on such adaptive aids as a "spork" (a combination spoon and fork) or the creative use of Velcro®. If there is a problem manipulating a button through buttonholes, buttons can be sewn onto Velcro. If a splint is needed, O.T. will provide it.

- The recreational therapist will help take the patient's mind off some of the rigors of physical therapy by such diversions as a shopping trip. Outings under the eye of the recreational therapist restore the patient's perspective that he or she continues to be part of the community beyond the hospital's confines. Want to go bowling? How about swimming or archery? Or a trip to see the Richmond Braves? All can be on the itinerary as the elderly person gains experience in getting around in public places again with crowds, curbs, and narrow stalls in public bathrooms. Then there can be camping. An 80-year-old patient was charmed and delighted when she went on her first camping trip; it was behind the hospital, but she nonetheless was out camping for the first time.

The recreational therapist also is involved in special activities within the hospital. Some recent ones at Sheltering Arms included a Hobo Day, in which the staff as well as the patients got into the spirit, a Hallowe'en celebration complete with a fun house, a T-shirt Day and a Hat Day. Ongoing programs include activities involving not only the patients but also their families, like family dinners. It's great for morale.

The therapists are part of a team that includes nurses, social worker, psychologist, pharmacist and chaplain who, together with the rehabilitation physician, seek to help make the elderly patient as mobile as possible.

People lose 3% of their strength daily when confined to a bed, and when this loss of strength is



complicated by a medical disability, such as hemiplegia, amputation or major surgery, the patient is made helpless. Appropriately selected patients given the proper support and correctly applied therapies are able to fight back in spite of advanced age.

The rehabilitation process is an active one. The patient is evaluated from the point of view of medical problems and disabilities as well as residual strengths. The patient is also evaluated in terms of social and physical environment. This uncovers the obstacles, and the evaluation is then presented to the patient along with a plan. In this way, a problem like a contracture of the hip inhibiting ambulation can be identified and restated as the goal of increasing the range of motion. If the problem is that the patient needs assistance in making a transfer from a wheelchair, the goal becomes independence in transferring. Even if the patient hopes to live with relatives after discharge from the rehabilitation hospital, the patient still needs the maximum degree of mobility within the home so that the others do not feel intolerably burdened.

Let goals be in reach, for they can then be stepped up. As the patient achieves a goal, the progress is emphasized. If progress is not proceeding as expected, a reason is sought, like infection, drug interaction, sleep disorder, depression or anemia. Medical treatment usually results in renewed progress in the various therapies.

The family as well as the patient must be thoroughly educated about all aspects of the care and health. They are taught the proper measures to try to avoid relapse. They are taught when to help, how to help—and when not to help.

The family is the unit of society, and its role in rehabilitation is the key to success. Many families are willing to provide shelter and assistance to their elderly members but are inhibited by ignorance of what is required and by fear of failure. Often this is compounded by a similar lack of understanding and fear on the part of their medical advisors.

In the medical rehabilitation model, contact is made with the family early and often. The doctor explains the patient's expected abilities following rehab efforts and seeks the family's support. Often, without the support of the family, rehabilitation efforts are likely to fail. If the goal of the program, for example, is to prevent nursing home placement

and the patient would have no other place to live, it cannot succeed.

As mobility and the ability for self-care increase, the patient is given more and more responsibility by being moved to a private room to practice the activities of daily living, getting therapy and meals without assistance, asking for medication at the appropriate time.

Preparation for discharge has already begun. An occupational therapist visits the home to check for access for a wheelchair, for example. The therapist will look for obstacles like scatter rugs and electrical cords. The bathroom is inspected. Recommendations are made.

The patient is then ready for therapeutic leave. That's an overnight pass during which Grandma and Grandpa have the opportunity to practice the skills they have learned at the hospital. They and their families are encouraged to define any difficulties they encounter at home. These problems become reshaped into goals when the patients return.

All of this helps create the well-rounded rehabilitation program, but the effort is wasted if the patient's independence doesn't continue after discharge. The rehabilitation program, therefore, must not only include developing hobbies and other avocational and social skills but also following up with a social and psychological as well as physical check up after one month. The patient is then either discharged to the care of the primary physician or seen regularly to supervise outpatient therapy.

The focus of the program is the worth of the individual, regardless of the age. Sometimes we're too quick to say, or at least to think, that a rehabilitation program for the elderly is directing money and the doctor's talents away from the young. The elderly, after all, frequently have a complicated set of intertwined medical problems and "have lived a full and useful life" already. At no time is the issue of cost-effectiveness more important than now, because if we are not cost-effective, we will not survive.

But I would rather emphasize the more important issue of quality of life. Rehabilitation is strongly committed to the dignity of mankind. Rehabilitation supports the individual's search for freedom, independence and self-determination. Grandma and Grandpa deserve no less.

# Home Care: A Boon to the Aged Ill

F. J. Spencer, MD, *Richmond, Virginia*

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Although much is made of the cost-effectiveness of home care, it is equally significant that the well-being of the patient can be enhanced by the familiar surroundings of home. Ultimately, the author observes, the home is the most desirable setting for the chronically ill, so many of whom are elderly.

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**I**N RESPONSE to today's climate of cost-consciousness, home-care service organizations are proliferating in Virginia at a great rate. Of the 245 requests for certificate of need applications received by the Department of Health in the year ending last July, 68 were for new home health projects.<sup>1</sup> To ease the burden on the bureaucracy, the General Assembly at its present session is considering exempting home health projects from the certificate of need process.

Home care is a particular boon to the aged ill; in many cases it can prevent institutionalization.<sup>2</sup> Options offered by today's home-care services include therapists, social workers, homemakers, Meals on Wheels, and helpers to relieve family members who care for invalids in their homes.

From the Department of Preventive Medicine, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Spencer at Box 212, MCV Station, Richmond, VA 23298.

## Definition and History

There are three, and only three, categories of patients—inpatients, outpatients, and home care patients. Anyone who needs care for 24 hours a day should be an inpatient in a hospital or nursing home; anyone who can attend an office or clinic should be an outpatient; the remainder should be home-care patients. Home health care, therefore, is for those patients who “cannot readily use outpatient services but do not require 24-hour institutional care”.<sup>3</sup>

Medical care in the home developed as part of the dispensary system of the 18th century. Prior to this, only the rich could afford care in the home. This was provided by a personal physician; the “Physician to the Queen's Household” still exists in England. The dispensaries “dispensed” medical care and medicine for the poor sick, and were the forerunners of hospital outpatient departments.

Recognition that many people could not attend a dispensary led to care being offered in the home. In this country, the Boston Dispensary established a home care program in 1796 to attend families in their own houses “at a less expense to the public



than in any hospital".<sup>4</sup> Thus home care was equated with less expense from the beginning. The Boston Dispensary's example was followed by visiting nurse agencies and public health departments.

Purists differentiate between "home care", a program providing services directed by a salaried physician responsible for the clinical management of the patients, and "home health care", a program caring for the patient in the home as an extension of hospital or office practice and supervised by the patient's physician.<sup>5</sup> In this paper, no distinction is made between these terms.

### Virginia Programs

As of February 1, 1984, there were 83 licensed home health agencies in Virginia, according to the Department of Health's Division of Medical and Nursing Facilities Services. Of these, 37 are operated by local health departments; 31 by commercial firms; nine by hospitals; and three by visiting nurse agencies in Arlington, Fairfax and Richmond.<sup>6</sup>

### Services

The basis of home health care is physician-directed nursing services. Additional disciplines commonly participating include physical, occupational and speech therapies; social work; and nutrition. Periodic review of the services supplied is required by Medicare and recommended in all cases, however financed. One neglected discipline is environmental health, often a contributing factor to the patient's comfort. Additional care is provided by home health aides and homemakers in many programs.

### Payment for Home Care

Financial support for home health services was originally quite different in health departments and visiting nurse agencies. Health Department services were supported by government funds and administered by government officials; those of visiting nurse agencies were funded by public donations and endowments, and supervised by volunteer boards of trustees. In 1966, with the advent of Medicare and, to a lesser extent, Medicaid, this distinction became blurred, and most care is now paid for by federal funds. This has led to commercial companies entering the field and to the introduction of licensure to prevent unhealthy competition and overcrowding.

### Licensure

All agencies providing care to patients in the home in Virginia must be licensed by the Virginia

Department of Health. The current rules and regulations for that licensure were approved by the State Board of Health in 1978. The requirements pertain to organization and management, program policies and procedures, records, physicians' plans of treatment, provision of services, and staffing requirements and duties. Annual relicensure is mandatory and is based on inspections of the service.

### Economics

Although the provision of health care in the home is less expensive than in an institution, *complete* home care is not necessarily cheap. Obviously, the cost of the services provided will vary with the condition of the patient, the number of visits required, and the range of services prescribed. The emphasis in any home care program is to teach the patient's family and friends to take care of the patient, thereby decreasing the overall cost of care.

### Patients and Diagnoses

Most patients cared for in the home are elderly, thereby qualifying them for Medicare payment. Heart disease, cancer, stroke and diabetes account for the majority of diagnoses encountered, and multiple pathology is common. Polypharmacy, or the prescription of multiple drugs, is also common and is often a source of bewilderment to patients who may already be confused by age and arterial disease.<sup>6</sup>

### Physician's Role

As the diagnostician and the prescriber of therapy, the physician must accept the responsibility of directing all aspects of care in the home. At first, this would seem to be an easy task, but if coordinated and continuing care is to be supplied, time is needed to do it well. Without due attention, however, the physician is entirely to blame if he or she cannot "keep control" of the patient—a complaint often voiced. The day may come, and rightly so, when group practices will have a "community physician" who coordinates all phases of patient care and watches trends in community services. Until a formally organized system of this type is realized, the practicing physician must be readily available to the agencies providing care, and review and revise plans of treatment periodically, whether required by law or not. Nothing is more infuriating to a home health nurse than to fume in frustration because she cannot discuss her patients with their physicians, or at least not until she has spent an inordinate amount of time and effort in this purely administrative aspect of her work.

Perhaps the main barrier to home care is the lack of incentive provided to the physician to incorporate it in his or her practice. This, of course, is not true for prepaid health plans, where the incentive to contain costs promotes the use of care in the home. One innovation is the return of the house call by practising physicians. This could lead to the formation of private practice home-health teams.

### Trends

With the advent of Medicare, providing home health services for profit became attractive. The result has been a burgeoning of home health agencies, although by no means all of them provide a complete range of services. Competition between agencies, although healthy up to a point, has produced duplication of services and, on occasion, a scale of fees tied to profit rather than service. Although the licensing regulations in Virginia control exploitation to some extent, there is still a lack of coordination of home health services in many areas, especially in the larger conurbations.

Ultimately, care in the home is the most desirable way of caring for the chronically ill. Apart from economy, the familiar surroundings of the home unquestionably contribute to the well-being of the patient. The acceptance of the principle of home care by hospitals can be applied to group practices and is already an integral part of many prepaid health plans. With the increase in the aging population, home health services would seem to be one of the most effective ways of providing care. It will probably expand in the remaining years of this century and beyond.

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## Alzheimer's aid is best seller

From the Johns Hopkins School of Medicine has come a handbook on Alzheimer's disease and other cognitive disorders in the elderly that has quietly become a best seller. Written by Dr. Peter Rabins, Nancy L. Mace and others of the school's Henry Phipps Psychiatric Clinic, it is titled "The 36-Hour Day: A Family Guide to Caring for Persons with Alzheimer's Disease, Related Dementing Illnesses, and Memory Loss in Later Life" and was offered to help the relatives of clinic patients cope with these diseases. Almost immediately, however, requests for the pamphlet began coming in from people unconnected with the clinic. When sales reached 200 copies a week, the authors revised and expanded the text to 71 pages, and the Hopkins University Press published it in 1982. At current monthly sales of 3,500, the paperback is in its ninth printing and is one of the ten top sellers in the press's 105-year history.

It may be ordered by sending \$6.95 plus \$1.50 postage to the Johns Hopkins University Press, Baltimore MD 21218.

## ABSTRACT

**Age Trends in Autopsy Rates. Striking Decline in Later Life.** Judith C. Ahronheim, MD, Alissa S. Bernholz, MPH, and William D. Clark, MA.

Age-related autopsy rates were computed from 99,145 death certificates and disclosed a striking decline with advancing age, the peak (82.5%) occurring in the third decade of life, the nadir (2.4%) by age 90 years. The trend was the same for both sexes, although at all ages men were more likely to undergo autopsies than women. There was a significant decline in late life for medical examiner (ME) and non-ME cases. Rates varied according to immediate cause of death. The only causes of death in which there was no significant age-associated decline were homicide, suicide, and transport accidents, where autopsy is mandated by law. For other causes of death analyzed, rates declined significantly with age in both sexes, for ME as well as for non-ME cases. We conclude that low autopsy rates in late life are a phenomenon deserving closer scrutiny. *JAMA* 1983;250:1182-1186



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# VIRGINIA MEDICAL

## EDITORIAL

### What Is a Geriatrician?

**T**HE WORD "geriatrics" was introduced by Dr. Ignatz Nasher of New York in about 1909. It comes from the Greek word *geras* + *iatics*, or pertaining to the physician, and refers to that branch of medical practice that deals with the care of the elderly just as pediatrics deals with the care of children. The great difference between these two disciplines is that Americans love their children, as their presence revives and renews hope for the future in the lives of parents and grandparents, while the older members of society seem to threaten the permanence of their kinfolks' lives and at times become financial burdens. In the beginning, four or five workers contributed to the Social Security system for one retired person; now it is down to about one in three and threatens further disparity between the total work force and the number of retirees. So the increased number of so-called senior citizens become a financial threat as well as a philosophical one.

No person would hesitate to have his child seen by a competent pediatrician, but many adults, even in their latter years, do not consider themselves old enough to consult a "geriatrician", if there is such a specialty.

Those physicians who are at least interested in treating the aged do not wish to be designated as geriatricians and, consequently, until recently there has been no movement to make geriatrics a bona

fide specialty. The medical schools of this country have been slow to recognize a growing need for younger physicians to become involved in the treatment of the aged. Fifteen years ago my medical school allotted one hour to a lecturer on geriatrics medicine; this was discontinued because it was stated "there is no room in the curriculum." Things have changed little for the medical student since then, although recently courses of gerontology are offered by the parent institution in its academic departments. Duke Hospital in North Carolina has a very large geriatrics and gerontological research operation and is to be commended for taking the lead in the South.

What is a geriatrician? He is a physician who loves, respects and admires old people as a starter. Perhaps he has to be born with this attitude, or at least taught at an early age. No matter how much technical skill he may acquire by education and clinical training and practice, he will not fulfill his role as a geriatrician unless he has this attitude.

Obviously, he must have an uncommon knowledge of human nature and of the dynamics of growing old, which, of course, increases as the years roll by in his own life.

Most young doctors naturally begin to build their practice with their peers in the society, at least in the primary-care field; but as their peers grow older, so do they, and most doctors who practice



more than 20 or 30 years acquire the parents of their peers and eventually the peers themselves in the field of caring for the aged.

However, the older doctors cannot possibly look after the increasing number of our fast-growing older population, which has already reached 11% of the total census.

It is true that for the young doctor the older patient may constitute a threat to his ego. He may feel that his training and medical school and through residency has enabled him to cope with any disease or solve most any problem, but when confronted with some geriatrics problems he can only act as a counselor and comforter to enable the patient to live with whatever disease he cannot cure.

Not only does the geriatric physician need a kindly attitude but a thorough knowledge of the dynamics of disease as it affects the aging body. He must become aware of the diminishing homeostasis and electrolyte balance and reaction to drugs, not only in quality but in quantity. He must also be aware of the diminishing reserves of the older patient in regard to his environment—heat, cold, stress, intake, output, rest, activity, etc. The old body is not as resilient as the young, and its recuperative powers neither as rapid nor as predictable.

The geriatrician, unlike the pediatrician, must realize that the 60- to 90-year-old not only presents himself or herself as a present patient but in the sum total of all the experienced illnesses and injuries, both physical and mental, plus the individual genetic pattern. These all have a bearing on the immediate disability for which he or she presents to the geriatrician. So in making a diagnosis and outlining treatment, the geriatrician must use his whole knowledge of internal medicine and his experience to solve the patient's problem.

In doing this, he must be able to separate the trivial complaints that frequently come with age but should be dealt with them in a kindly way from the more serious signs and symptoms which may not even bother the patient but raise a red flag of diagnostic importance in the mind of the doctor.

Great patience is needed in dealing with the aged. Many move slowly, dress and undress slowly, come late for their appointments, or come on the wrong day, the wrong week, the wrong month, but expect to be seen whenever they arrive and quickly. The more time they have at their disposal, the more they expect immediate service from their doctors.

Many say they don't want to live much longer. Most are not afraid to die. They will tell their doctors not to prolong their existence if they have

an incurable terminal illness. The role of the geriatrician is that of not necessarily extending their life span but of keeping them comfortable while they live.

The geriatrician must enter into advice about the patient's life-style as he or she grows more dependent on others for environmental support. Should the patient continue to live alone or with family, or some alternative living arrangement, whether it be in the retirement home, adult home or, if conditions of health are severe enough, a nursing home?

The family, of course, enters into such decisions, if the patient has a family. Most families have a guilt feeling when mother or father reach the stage when the facility becomes necessary. Unfortunately, in our society older people have exacted promises from their children that they never permit their admission to a nursing home. However, in Alzheimer's disease this simply becomes necessary, for the patient has lost the ability to cooperate or to reason as to what is best. Most families see the wisdom of the physician's strong advice and accept it. So the geriatrician may have to play the role not only of physician but that of social worker, counselor, even pastor, but most of all a true friend indeed.

The care of the terminally ill patient presents a challenge to the geriatrician. How far should one go to differentiate between the comfort of the patient on one hand and on the other hand extend his life span? When a patient can't swallow, should a nasogastric feeding be introduced, and if so, for how long? If the patient can't breathe unassisted, how long is a mechanical respirator indicated? If these measures result in eventual return of function, well and good, but if these measures only prolong the agony of dying, then they should be terminated. We don't need government intervention to hamper our clinical judgement any more than it is needed in the so-called "Baby Doe" situation. Terminal care should be left to the doctors, not to the politicians.

The prayer of the geriatrician, then, has to be one so aptly stated by Reinhold Niebur: "God give me the serenity to accept the things I cannot change, the courage to change the things I can, and the wisdom to know the difference." A geriatrician must have that wisdom if he is to perform his duty and survive himself.

JOHN P. LYNCH, MD

McGuire Clinic  
7702 Parham Road, Richmond, VA 23229

Dr. Lynch is a member of the Committee on Aging of The Medical Society of Virginia.

# VIRGINIA MEDICAL OBITUARY

## Frank H. Thaler, MD

Dr. Frank H. Thaler, McLean psychiatrist, died after a long illness July 1, 1983, at Sibley Memorial Hospital in Washington. He was 49 years old.

A native of New York City, Dr. Thaler was graduated from the Newark, New Jersey, College of Engineering in 1956 and for 14 years was an engineer with the RCA Corporation in New Jersey. He earned his medical degree in 1976, at the Chicago Medical School, and trained in psychiatry at Georgetown University Hospital, Washington.

Dr. Thaler was a member of The Medical Society of Virginia, Fairfax County Medical Society and the American Psychiatric Association.

## Memoir of J. R. Beckwith 1910–1982

John A. Owen, Jr., MD,  
Lockhart B. McGuire, MD,  
and John L. Guerrant, MD

Julian Ruffin Beckwith, Jr., MD, died at his home near Ivy, Virginia, on September 17, 1982, at the age of 71.

He was born December 28, 1910, in Petersburg, the son of Julian R. Beckwith, MD, and Louise C. Beckwith. Educated in Petersburg public schools, he attended Virginia Polytechnic Institute and the University of Virginia School of Medicine, receiving his MD degree in 1936. After a rotating internship at the Cincinnati General Hospital, he returned to the university and spent several years in medical residency and fellowship before moving in 1940 to the C&O Hospital in Clifton Forge, where he soon became chief of medicine. Except for a military leave of absence from 1943–1946 (research service as captain in the Army Medical Corps), he spent 13 productive years at Clifton Forge, expanding the clinical facilities in internal medicine there, becoming the preeminent diagnostician and cardiologist in Western Virginia, and publishing a dozen excellent research papers without any institutional research support.

He returned to the University in 1953 as assistant professor of medicine in cardiology under the late J. Edwin Wood, Jr. By 1965 he had risen to the rank of

professor and head of the Cardiovascular Division, a position he held until 1977. In 1981 he retired as emeritus professor of medicine but continued active practice as a consultant until the onset of his final illness in July 1982.

Over the last 30 years, Julian Beckwith's career epitomized excellence in clinical cardiology. In addition to the scholastic honors of membership in AOA, the Raven Society, Sigma Xi and ODK, he was a fellow of the American College of Cardiology; a member of the American Heart Association, the American Clinical and Climatological Association and the Association of University Cardiologists; and president of the Virginia Heart Association for many years. He was always an ardent supporter of the American College of Physicians, serving as governor for Virginia from 1966–72.

During his academic career he produced 54 research and clinical publications, a revision of Grant's *Clinical Electrocardiography*, and his magnum opus, *Basic Electrocardiography and Vectorcardiography*, which appeared posthumously. His skills in patient care and administration were matched by those in teaching: twice, in 1970 and 1981, he received the Robley Dunglison Award as the outstanding clinical teacher at the University of Virginia School of Medicine. The American College of Physicians honored him posthumously with mastership, the College's highest award.

His colleagues remember Dr. Beckwith as a man jaunty and confident even in the face of calamity, a delightful friend and companion, a superb teacher, and one who always delighted in working with the house staff. Other attending physicians might grumble and delay if asked to make rounds with the residents; Julian Beckwith was always available, with the proud smile, "It would be an honor!"

In 1936 he married Lois Hughes of Lynchburg, a graduate of the University of Virginia School of Nursing. Their family includes four children: Julian R. Beckwith, III, PhD, of Athens, Georgia; Lois B. Johnson of Ann Arbor, Michigan; George H. Beckwith, MD, of New Bern, North Carolina; Polly B. Hawkes of Hershey, Pennsylvania; and ten grandchildren, including Julian Beckwith IV and Julian Beckwith Johnson.

Aside from his love of medicine and his family, Julian's boundless enthusiasm went into his pursuit



of the ancient Virginia sport of fox-hunting. Mounted on his favorite brown mare (known as "Consultation" to envious colleagues and as "Panacea" to his understanding family), he was a familiar and energetic figure at the Farmington Hunt Club, which he served as president for four years, 1973-1977. With his instinctive loyalty to all things from the Old South, he called to mind Jeb Stuart's cavaliers, riding clean around McClellan to the tune of "If You Want to Have a Good Time, Jine the Cavalry."

When in July of 1982 the diagnosis of his fatal illness became known, Julian accepted it with undaunted equanimity that awed his sorrowing colleagues. Retiring to his home in western Albemarle County, he gathered friends and family around him, teasing and reminiscing, charming and encouraging them, as if he were the physician and not the patient. Never was the art of management of the dying patient so beautifully personified in one fearless man, whose down-to-earth humor and courage made him truly the master of his own fate. And at the end he was "like one who wraps the draperies of his couch about him and lies down to pleasant dreams."

He was buried in the cemetery of St. Paul's Episcopal Church in Ivy, where he had long been a faithful member. His memory will be preserved not only by the recently established Julian R. Beckwith Professorship of Medicine, but by the indelible stamp of love, integrity, and pride that his life has imprinted on all who were privileged to know him.

## Memoir of James L. Davis, Jr. 1912-1983

*By the Rev. Ralph M. Piland, Bliss K. Weems, MD,  
and J. Powell Anderson, MD*

Dr. James Lucius Davis, beloved Waynesboro family physician for 40 years, died January 2, 1983. He had retired from Associated Physicians, Inc., in June of 1980 for health reasons.

Born to James Lucius and Elisabeth Bryant Crittenden Davis on January 19, 1912, in Orange County, Virginia, where a part of the Civil War was fought, he lived out his boyhood days on the battlefields and in the shadows of that tragic con-

flict. The silent but real companions of his youth were the heroes of that bit of history. He shared more than the same birthday of January 19 with the legendary Robert E. Lee. He shared Lee's passion for integrity and honesty and devotion to duty.

Dr. Davis was educated at Virginia Polytechnic Institute and the University of Virginia School of Medicine, where he graduated in 1936. Following an internship of two years in the state of New Jersey, he began his medical practice in the West Virginia and Kentucky coal mining country before coming to Waynesboro in 1941 to practice in partnership with Drs. D. Edward Watkins, James J. Hubbard, and Bliss K. Weems. From 1942 to 1946 he served in the United States Army Air Corps, with overseas duty in the African theater. He resumed his Waynesboro practice in 1946.

Dr. Davis belonged to the usual professional organizations, but it was his broader kinship with, and service to, humankind that set him apart as someone special. In 1982, the Waynesboro Exchange Club sought to pay tribute to this man of uncommon stature by making him the recipient of its "Book of Golden Deeds" award. About two decades ago, Dr. Davis lost the ability to walk without the aid of braces, yet it was a common sight to see him making a house call late at night in the most severe weather even though sometimes he literally had to drag himself up the steps.

He was a gifted man and had a keen interest in areas other than medicine. He had a love affair with God's creation and was a responsible outdoorsman. As long as he was able to walk, he was an enthusiastic hunter. And he remained a fisherman par excellence almost until the time of his death.

No one pays adequate tribute to Jim Davis without mentioning his basic character. He was a quiet and unassuming man, even shy. But a rapier eloquence would flow from his tongue when he felt that anyone had violated good medical ethics or becoming professional conduct. At heart he was gentle and kind and was constantly befriending the down-and-out of the community. That's why he was so loved and will be long remembered.

He is survived by his wife, Katherine Longcor Davis, and one son, Dr. James L. Davis III, a pathologist in Seal Beach, California.

Prepared and published at the request of the Augusta County Medical Society.

# WHO'S WHO

After 33 years of service to Giles County patients, **Dr. Lester E. Dunman** of Pearisburg has closed his practice. His retirement leaves a hole not only in the medical community but in civic affairs as well, for he is identified with school board service; with local fund drives, such as those for the American Heart Association and Cancer Society; with the Lions' Club, of which he has been president; and perhaps most of all with the town's recreation program, for it was he who supervised the construction of the Pearisburg swimming pool.

Born in West Virginia, Dr. Dunman was graduated from Virginia Tech and the Medical College of Virginia. After a stint as an army doctor and a tour of duty in France, he completed his training in Pittsburgh, Pennsylvania, then headed for Pearisburg with his family in 1930 when he heard that Giles County was looking for doctors. He and his wife have four children, all now living in other states, and 11 grandchildren.

Long a member of the Giles Memorial Hospital's medical staff, Dr. Dunman has been chairman of its Continuing Education Committee and was recently honored by having the hospital's intensive care unit dedicated to him. And recognition on his retirement was recorded by the Pearisburg *Virginia-Leader* in an article by Caroline Kane from which this information was taken.

These four Medical Society of Virginia members have been elected to fellowship in the American College of Physicians: **Dr. Winston M. Ueno**, Alexandria; **Dr. Furadoon A. Irani**, Front Royal; **Dr. Russell D.**

**Evett**, Norfolk; and **Dr. Michael P. Higgins**, Virginia Beach.

**Dr. Cary N. Moon, Jr.**, Kilmarnock otologist, was elected president of the American Otological Society at its annual meeting in New Orleans. Dr. Moon has served the 200-member organization in progressively responsible posts since 1973. He is a graduate of the University of Virginia School of Medicine, where he also trained and is now a clinical professor.

No sooner had **Dr. Harold L. Williams** completed his term as President of The Medical Society of

Virginia than he consented to lead as chairman the Virginia Medical Political Action Committee. Serving with the Newport News surgeon are **Dr. Gerald C. Burnett**, South Boston, chairman elect; **Dr. Barbara A. Mella**, Fairfax, vice chairman; **Dr. H. Alan Bigley, Jr.**, Petersburg, secretary; and **Dr. John A. Rawls**, Richmond, treasurer.

On **Dr. Alexander McCausland**, Roanoke, was conferred the Distinguished Clinical Award of the American Association of Clinical Immunology and Allergy when it met late last year in Kissimmee, Florida. Dr. McCausland is a past president of both the association and The Medical Society of Virginia.

Gov. Charles S. Robb has reappointed **Dr. Robert J. Faulconer**, Norfolk, to the board of visitors of the College of William and Mary.



**Grandpa Dunman with Scott Koehler, one of the eleven.**

*Photo by Caroline Kane for the Virginian-Leader*



**Dr. William J. Kerns**, Front Royal, has been named a fellow of the American Academy of Family Physicians, and **Dr. Isam Zibdeh**, Norton, has been elected to fellowship in the American College of Obstetricians and Gynecologists.

Elected secretary of the American Society of Anesthesiologists at its annual meeting in Atlanta was **Dr. Randolph Mott Jackson**, Winchester, who was also named a delegate to the 8th World Congress of the World Federations Society of Anesthesia held late in January in Manila, the Phillipines. Dr. Jackson is a past president of the Virginia Society of Anesthesiologists.

**Dr. Robert W. Cantrell**, Charlottesville, was reelected southern vice president of the American Academy of Facial Plastic and Reconstructive Surgery when its members met in Anaheim, California, recently. Chairman of the Department of Otolaryngology and Maxillofacial Surgery at the University of Virginia, Dr. Cantrell also heads the university's new Navy reserve unit, established by the US Department of Defense to give physicians the training needed to respond to a national emergency. Dr. Cantrell was on active duty with the Navy for 16 years.

**Dr. Donald Barry Nolan**, Roanoke neurologist, has been elected to the board of directors of Blue Cross and Blue Shield of Virginia. Dr. Nolan is president of the Southwestern Virginia Medical Society and president elect of the Virginia Neurological Society.

For his key role in reviving interest in needle aspiration biopsy for the diagnosis of cancer, **Dr. William J. Frable**, Richmond, has been given the George N. Papnicolaou Award of the American Society of Cytology. Dr. Frable, pathologist at Rich-

mond Eye and Ear Hospital, was president of the society in 1979.

AS VIRGINIA MEDICAL reported in its January issue, **Dr. Wilson C. Merchant III** and **Dr. W. S. Turner**, Richmond, won first prize for their scientific exhibit on "Pediatric Blunt Renal Trauma" at The Medical Society of Virginia's annual meeting at the Homestead, but notice of the second and third prize winners was, regrettably, not included.

Second prize was awarded to two other Richmond physicians, **Dr. Jaime Tisnado** and **Dr. Shao Ru Cho**, for an exhibit titled "Percutaneous Unknotting and Retrieval of Intravascular Catheters and Other Iatrogenic Foreign Bodies."

To **Dr. Esrafil Abedi** and **Dr. Mary Ann Frable**, also of Richmond, went third prize for their exhibit on "Repair of Severe Laryngeal Stenosis."

For his contributions to his alma mater, his church and his community, **Dr. Percy Wootton**, Richmond cardiologist, was given the 1983 Thomas Gibson Hobbs Memorial Award of Lynchburg College. The award is presented each year by the college's Alumni Association. A member of Lynchburg College's Class of 1953, Dr. Wootton is a trustee of the college.

As a medical student in the early 1960s at Nishtar Medical College in Multan, Pakistan, **Dr. Khalid J. Awan** studied under Professor M. Shafi, founder of the college's ophthalmology department. Twenty years later Dr. Awan, now an ophthalmologist in Norton, Virginia, travelled to Multan to deliver the college's Shafi Memorial Lecture. Setting for the lecture was the International Conference on the Advances in Medical Sciences sponsored in December 1983 by Multan University; the president of Paki-

stan himself, Gen. M. Zia-ul-Haq, inaugurated the conference. Dr. Awan's topic was "Angoid Streaks of the Ocular Fundus: Their Significance in Systemic and Local Diseases."

Appointed acting dean of the Medical College of Virginia to fill the vacancy created when **Dr. Jesse L. Steinfeld** left for the Medical College of Georgia was **Dr. Leo J. Dunn**, chairman of the Department of Ob-Gyn. He will serve for at least a year.

The 17 years of service of **Dr. Monte Binder**, Newport News, was honored by the Virginia Affiliate, American Heart Association, when he was given the organization's distinguished achievement medallion. Dr. Binder won the medallion once before, in 1974.

**Dr. Shaun Ruddy**, Richmond, was the recipient of the Distinguished Achievement in Research Award at a Virginia Commonwealth University convocations program honoring distinguished faculty members. He is chairman of the Division of Immunology and Connective Tissue Diseases.

At the University of Virginia, **Dr. Richard F. Edlich** has been named Distinguished Professor of Plastic Surgery, and **Dr. John Staige Davis IV** was elected the Margaret M. Trolinger Professor of Rheumatology.

**Dr. Charles J. Goldman**, Norfolk, is the new president of the DePaul Hospital medical staff, and in Suffolk, **Dr. Douglas U. Kells** was elected president of the medical staff of Louise Obici Memorial Hospital.

In Staunton, **Dr. Mardre Bell** was elected president of the medical staff of King's Daughters' Hospital.

# VIRGINIA MEDICAL CLASSIFIED

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## NEW MEMBERSHIP DIRECTORY COMING

The second edition of Virginia Medical's Directory of Medical Society of Virginia members will come to you in the August 1984 issue. Your name and address will appear as shown on your mailing label, which you will find on the back cover of this issue. Is it correct? If not, send your revisions to Virginia Medical, 4205 Dover Road, Richmond VA 23221.

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# BOOKS

**Systems Approach to Emergency Medical Care**, by David R. Boyd, Richard F. Edlich and Sylvia Micik. East Norwalk Connecticut, Appleton-Century-Crofts, 1983, 528 pages, illustrated, \$46.50.

THIS TEXTBOOK will be valuable to any physician, administrator or related professional interested in emergency medical care. As noted in the preface, the text, "a real first" related to this area, reviews the experience obtained through the emergency medical services development in the United States over the past decade. Target areas of care, assessment, objectives, organization, education, implementation, clinical care and evaluation of various emergency medical systems are integral aspects of this work.

Interestingly, each of the 19 chapters is a monograph which could stand alone, yet the common themes of organization, education of health care providers as well as the public, optimal achievable patient care, program review and dynamic systems for creating emergency medical facilities are stressed in each. The text consists of two parts, the first devoted to the development, organization and implementation of emergency medical services, and the second to discussion of individual clinical emergency problems. The authors of the chapters are recognized authorities in their respective areas.

The initial chapter is a precise documentary of the history of emergency medical services (EMS), dating from the Civil War in 1861, past the first automobile accident in 1885, to the beginning of the emergency medical systems in 1966 as addressed in a document from the National Academy of Sciences, National Research Council. Though somewhat tedious for the clinician, the chapter will be a valuable record as emergency medical services evolve in the future.

The organization, administration and management of an emergency medical service is then adeptly presented. Agency characteristics, job descriptions, government agencies' and professional organizations' involvement, and community and political supports are well defined. Next the training of emergency medical services personnel is re-

viewed, with a presentation of proven educational concepts of curriculum and objectives. Medical control and accountability, the overall medical direction and the role of paraprofessional personnel is then explained. The pre-hospital transportation of patients by ground and air is considered, with emphasis on training personnel, costs, evaluation, response times, organization and quality control. An appendix of recommended medical equipment for the aero-medical craft is included. Evaluation of the emergency medical service is discussed as an ongoing process, with quality control of data collection emphasized. The first section concludes with advice for the emergency services medical director who might become involved in the legislative process; this chapter includes a model state EMS bill which will be helpful for those just beginning an EMS program.

The remaining chapters address individual clinical emergencies, including psychiatric, burns, cardiac, high-risk infant, head, spinal cord, poisoning, radiation, reimplantation and trauma. For each problem the emergencies are categorized and severity of injury is frequently outlined. The pre-hospital management, transportation and hospital care are elucidated. Some chapters deal with patient care in more depth than others. Each chapter treats the emergency system on a regional basis, stressing individual organization of the initial response, training of pre-hospital personnel.

The text closes with a chapter on trauma, with the system from roadside care to all levels of subsequent care described in detail and a review of the relevant published literature.

The book contains clear and well-explained figures and tables and helpful appendices. There are a few well-chosen pictures, including some excellent ones in the chapters on spinal cord injury and reimplantation.

This textbook will serve as a valuable document for beginning or improving the EMS in your community, and it will be an invaluable aid to anyone interested in emergency medical systems. Had this fine book been available at an earlier time, the emergency medical system of the nation might well be in a more advanced stage than it is. The editors have accomplished their objective of ensuring that emergency medical systems will no longer be considered the "neglected" area of our nation's health and safety.

ROBERT E. BERRY, MD

Roanoke Memorial Hospitals  
Box 13367  
Roanoke VA 24033



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## It's spring and the HMOs are sprouting

HEALTH MAINTENANCE ORGANIZATIONS are springing up all over Virginia. Richmond is a hotbed, with three new HMOs taking root, but in the Tidewater area, new plans are bustin' out all over, and in Northern Virginia a new HMO has begun harvesting subscribers.

**In Richmond**, the new HMO likely to come to fruition first is St. Mary's Hospital Association. Organized by physicians who practice at the non-profit St. Mary's Hospital in Richmond's West End, this plan allies the physicians, the hospital, and Blue Cross Blue Shield of Southwestern Virginia, which is to begin marketing the plan this month.

*In its November 1980 issue, VIRGINIA MEDICAL published its "Guide to HMO-Watching," with Gary Brookins' HMO bird on the cover. Here is another in the series of followup articles.*

Dr. Yale H. Zimberg, thoracic and cardiovascular surgeon, is president of the IPA-type association.

There are 275 physicians on the active staff at St. Mary's. All may apply to contract with the new association, and by early last month the applications of about 125 had been approved.

Why the hook-up with the Roanoke-based Blues in Richmond, where BC/BS of Virginia is located?

The Virginia legislature has dismantled the geographical boundaries that used to limit the two companies' sales reps; now they are competing for subscribers throughout the state, and the Roanoke firm "offered us a better deal," Dr. Zimberg said.

Scheduled to begin enrolling subscribers only a month after the St. Mary's start-up is United Medical Plan of Virginia, Inc., the enterprise of Lawrence N. Kugelman and Roger S. Taylor, MD, both of whom formerly worked for an HMO in Southern California called INA and Ross Loos Healthplans. (Dr. Donald Ross and Dr. H. Clifford Loos pioneered the prepayment scene in California beginning in 1929. INA signifies Insurance Corporation of North America.)

Kugelman heads up United Medical as president, and one of the first things he did was hire as the plan's associate medical director Dr. Edward A. Zakaib of Richmond. Dr. Zakaib, a family physician, is chairman of the Medicine/Business Coalition Committee of The Medical Society of Virginia.

As of early March, this new HMO has signed up 250 Richmond physicians and was in the final stages of contracting with "a number of hospitals," according to Kugelman. Tentative overtures had been made toward several big employers in the area, with the goal of enrolling subscribers beginning the first of May.

Is there room in Richmond for all these plans? Kugelman, who is used to the teeming HMO scene on the West Coast, is confident there





# NEWS BUREAU

is. He and his associates studied 30 cities for a location for United Medical Plan, he says, and settled on Richmond because of the receptivity they found in both employers and physicians.

The third new contender in the capital city will be the Richmond connection of a statewide HMO network being staked out by Blue Cross Blue Shield of Virginia. HMO-watchers say that 20% of all Virginians will be enrolled in HMOs by a decade hence; a web of HMOs across the state could capture quite a few of them. To develop the network, the Virginia Blues have set up a non-profit company, Virginia Health Maintenance Organization, Inc., as a wholly owned subsidiary.

The network's Richmond component is to be activated early in 1985, according to a spokesman for the carrier. Individual contracts are to be negotiated with a number of solo practitioners and MD groups in the Richmond area, creating an HMO/IPA.

As news of these plans hit the headlines, a group of Richmond physicians quietly continued to explore the feasibility of setting up an independent, physician-owned HMO/IPA. They have incorporated as the Southern Health Management Corporation under the leadership of these officers: Dr. Emerson D. Farley, Jr., president; Dr. Ronald K. Davis, vice president; Dr. John M. Daniel III, secretary; and Dr. Edward M. Saylor, treasurer.

The seeds of this project were sown a year ago when Dr. Davis, then president of the Richmond Academy of Medicine, appointed a task force headed by Dr. Farley to investigate competitive alternatives

## Two more join the Tidewater competition

As these pages were going to press, two more plans announced they would join the contract medicine crowd in the Tidewater area. One is a PPO, one an HMO.

The PPO is to be the creation of three Tidewater hospitals: DePaul Hospital of Norfolk and the General Hospitals of Chesapeake and Virginia Beach, with a total of 811 beds.

And owners of United Medical Plan of Virginia, Richmond's newest HMO, announced they would set up a second shop in the Tidewater area.

The tally as of March 24 of contract plans now operating in Virginia or publicly committed to do so:

**In Richmond:** St. Mary's Hospital Association (IPA); United Medical Plan of Virginia (HMO); KeyCare (PPO); PruCare (HMO); Virginia Health Maintenance Organization network (IPA).

**In Tidewater:** Health Plan of Virginia (IPA); IPA of Southeast Virginia; KeyCare (PPO); BC/BS network (HMO); PPO of three hospitals; United Medical Plan of Virginia (HMO).

**In Northern Virginia:** Virginia Health Plan (HMO).

to Blue Cross Blue Shield of Virginia's new preferred provider organization, but ties with the Academy have long since been severed.

On hand to greet any and all of these challengers is the Blues' PPO, KeyCare, and Richmond's first HMO, PruCare, which opened for business late in 1982 with physicians of the McGuire Clinic under contract for medical service. Both plans are said to be flourishing. And of course the Blues' traditional coverage continues.

**In the Tidewater area,** Blue Cross Blue Shield of Virginia is scheduled to set up two new alternate delivery routes this year. First, on July 1, it is to open a Norfolk-based extension of KeyCare. Second, on October 1, the Norfolk component of the statewide HMO network is to start up.

Unlike its IPA-style counterpart in Richmond, the Norfolk stop on the

Blues' network is to be an HMO of the closed panel type. Signed up to provide care for its subscribers is the Norfolk Diagnostic Clinic. The clinic's staff of 28 internists offers a comprehensive range of subspecialties under the leadership of Dr. Stuart B. Baker, medical director.

Continuing to evolve in Norfolk is an HMO/IPA originated by physicians, Health Plan of Virginia, Inc., which has designs on subscribers not only in Norfolk but in the neighboring cities of Portsmouth, Virginia Beach and Chesapeake.

Health Plan of Virginia had its genesis late last summer and as of early last month had signed up 250 doctors, all of whom bought stock in the corporation. Seven business firms and five hospitals have invested in the plan, including the 450-bed De Paul Hospital.

Directing this plan's growth are two pros, Timothy Driskel, who was

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formerly with an HMO/IPA in Oregon, and Harold Green, who formerly worked in Washington for the Office of HMOs, Department of Health and Human Services.

The plan's board of directors includes 31 physicians and ten persons representing the business community and the consuming public. Officers are Dr. Claude A. Smith, president; Dr. Patrick C. Devine, Sr., vice president; and Dr. Leonard L. Davis, Jr., secretary.

Gearing up to enter the alternative delivery fray is a Tidewater giant, Medical Center Hospitals, which is comprised of the 250-bed Leigh Memorial Hospital and the 700-bed Norfolk General Hospital.

Norfolk General is one of the wonders of the Tidewater world. An enormous structure nine stories high and encompassing a city block, it is "constantly adding new wings," as one Norfolk doctor put it; construction was recently begun on 20 operating rooms. Physically connected to the adjoining Children's Hospital of the King's Daughters, Norfolk General is the teaching hospital for Eastern Virginia Medical School.

The executive management of Medical Center Hospitals reports to Alliance Health Systems, a not-for-profit holding company that owns, among other things, four free-standing emergency centers. Norfolk physicians have not taken kindly to this competition, and early this year they were further abridged when Alliance executives announced their intent to form an IPA-type partnership between the two hospitals and the single MCH medical staff.

There are 600 physicians on that medical staff. The president is Dr. William P. Edmondson, Jr. Dr. Edmondson is one of the internists of the Norfolk Diagnostic Clinic, the clinic that has signed up with the Virginia Blues' HMO network.

The medical staff appointed a committee to research and develop its role in the partnership. When a lawyer advised incorporation, the "IPA of Southeastern Virginia" was formed, and the half dozen doctors who agreed to stand in as incorporators elected Dr. Robert A. Morton as chairman. Long identified with PSRO activities in Virginia, Dr. Morton is pro tem chairman of the board of the new Medical Society of Virginia Review Organization.

Insiders say that the medical staff's initial reaction to the proposed partnership ranged from chilly to hostile, but they believe that attitude is changing. In any event, the committee's work was "moving right along," Dr. Morton said, when VIRGINIA MEDICAL's reporter talked to him, and a report may be ready this month.

Participation in the plan is to be open to any physician on the staff at a "minimal" investment, Dr. Morton predicted. The plan may be operative by summer, although he considers later likelier.

**In Northern Virginia**, the fledgling Virginia Health Plan counted 170 physicians signed up as providers and a beginning subscriber roster of seven firms when it became operational as an HMO/IPA late in February. Based in Fairfax, it is the sixth HMO in the Washington area but the only one with headquarters in Virginia, although Kaiser-Georgetown, the closed-panel HMO owned by Kaiser-Permanente, has facilities on the Virginia side of the Potomac.

Virginia Health Plan intends to deliver service in Arlington, Fairfax and Prince William counties and the cities of Alexandria, Falls Church, Fairfax and Manassas. Its target is business groups with a minimum of 25 workers. The monthly premium is about \$76 for individuals and \$190 for families,

although rates vary according to the characteristics of the subscribing group.

This plan is the enterprise of the National Hospital Health System Corporation, a holding company that owns the National Hospital for Orthopedics and Rehabilitation in Arlington. Dr. John C. Bucur, who is chief of neurological surgery at the hospital is chairman of Virginia Health Plan's board of directors.

**Other alternatives:** Two more ambulatory surgery centers are on the way in Virginia. Latest to receive the requisite certificates of need are Winchester Memorial Hospital and the University of Virginia Medical Center, bringing the state's ASC total to five. The other three belong to St. Mary's Hospital, Richmond, whose new HMO/IPA is reported above; the Lewis-Gale Clinic in Roanoke; and a group of Richmond urologists headed by Dr. C. M. Kinloch Nelson. Dr. Nelson is the "CON artist" of the feature story by that name in VIRGINIA MEDICAL's November 1983 issue.

The "Surgi-Center" in Winchester is to be built on a 94-acre suburban site to which the hospital hopes some day to transfer the entire facility. Dr. Richard L. Fieo is president of the ASC's board. Dr. Randolph Jackson is medical director.

At the University of Virginia, the freestanding ASC will be, at the outset, one story high and four ORs wide. It will cost \$1.6 million—peanuts compared to the \$119 million price tag of the university's 445-bed replacement hospital, on which construction is to begin this fall. And coming soon to Charlottesville to delight Dr. Jay Y. Gillenwater, chairman of the urology department, is a lithotripter ("stone crusher"), the first example of this heralded high-tech in Virginia.

—ANN GRAY

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# WHO'S WHO IN VIRGINIA MEDICINE

The Louise Obici Memorial Hospital's annual award for outstanding contributions to the medical profession was shared this year by two Suffolk physicians, **Dr. James M. Habel, Jr.**, obstetrician-gynecologist who retired last year, and **Dr. W. Holmes Chapman, Jr.**, internist. Both are long-time members of the hospital's medical staff. They were to receive the award at the hospital's clinical conference early this month in Suffolk.

It is fitting that **Dr. Walter A. Eskridge** of Parksley was co-chairman of a heart fund drive on the Eastern Shore last month, as the *Eastern Shore News* noted, because Dr. Eskridge had by-pass surgery at the Medical College of Virginia a few years ago—and six weeks later was back looking after his patients. Dr. Eskridge heads up the Eastern Shore Chapter of the American Heart Association, whose funds have nurtured bypass research.

New president of the Neurological Society of the Virginias is **Dr. Darwin J. Ferry, Jr.**, Roanoke, who took office at the Society's annual meeting in January in Williamsburg. Serving with him are **Dr. Robert P. Singer**, Richmond, president elect, and **Dr. Jacques E. Botton**, Lynchburg, secretary. The Society's 1984 Crutchfield, Gage and Thompson Award for the best paper presented by a resident was given to Dr. Dennis G. Vollmer of

the University of Virginia's neurological staff; his topic was "The Effect of Intraarterial Acetylcholine, Adenosine and Dipyridamole on Cerebral Blood Flow."

New president of the Virginia Dermatological Society is **Dr. Gerald C. Burnett**, South Boston. The vice president is **Dr. L. William Kelly**, Richmond, and **Dr. David M. Pariser**, Norfolk, is secretary-treasurer.

When the American Cancer Society, Virginia Division, held its 46th annual meeting in Reston, **Dr. Lloyd B. Burk, Jr.**, Arlington, was given the Terese Lasser Award for his outstanding contributions to the Reach to Recovery program.

**Dr. Arthur J. Martin**, Bowling Green, has retired after 32 years as a general practitioner serving the citizens of Caroline County. He relinquished his practice to **Dr. Edward B. Beirne**, although he will continue to call on a few patients under long-term care.

Born in Presque Isle, Maine, Dr. Martin came to medicine by a roundabout route, according to a feature story about his retirement in the *Caroline Progress*. After graduating from high school at age 16, he moved to New York City to live with his brothers and there found work as a jazz guitarist with the bands that played at charity balls and prep school socials. But World War II interrupted his musical career, and a tour of Army duty in Richmond led to his return to Virginia after the war to enroll in the University of Richmond and the Medical College of Virginia. He was graduated from MCV at age 40 and soon afterwards began his practice in Bowling Green.

Dr. Martin married Frances Sheffield of Richmond, and both Mrs. Martin and their only child, Peggy, have worked in the doctor's office.

"It's been a real family practice," Dr. Martin observes. He hopes now to travel with his wife and to get back to his music.

Under the editorship of **Dr. Lazar J. Greenfield**, chairman of the Department of Surgery at the Medical College of Virginia, members of the department have collaborated with authors across the country to produce *Complications of Surgery and Trauma*, a textbook recently released by J. B. Lippincott Company. Of the book's 67 chapters, 44 were written by faculty and staff at MCV.

New chief of staff at Stuart Circle Hospital, Richmond, is **Dr. Fleming W. Gill**, general practitioner in Richmond.

At Alexandria Hospital, **Dr. David A. Bernanke**, Alexandria internist, is the new president of the 600-member medical staff.

New chief of staff at Johnston Memorial Hospital in Abingdon is **Dr. William A. Nuckols**, family practitioner.

In Richmond, **Dr. Dennis A. J. Morcy**, internist and gastroenterologist, was elected chief of staff at Henrico Doctors Hospital.

At Winchester Memorial Hospital, **Dr. John E. McAllister** of Winchester was elected president of the medical staff. He is a neurosurgeon.

**Dr. Mamerto B. Adrales**, general surgeon in Covington, is the new chief of staff of Alleghany Regional Hospital, Covington.

Elected chief of staff for 1984 at Montgomery County Hospital, Blacksburg, was **Dr. Alex A. Tan**. He is a general surgeon in Blacksburg.

# An added complication... in the treatment of bacterial bronchitis\*

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**Contraindication:** Cefaclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

**Warnings:** IN PENICILLIN SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS. AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Cefaclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

**Precautions:** General Precautions—If an allergic reaction to Cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antioglobulin tests are performed on the mother's side or Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Cefaclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Cefaclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clintest\* tablets but not with Tes-Tape\* (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

**Usage in Pregnancy—Pregnancy Category B**—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Cefaclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

**Nursing Mothers**—Small amounts of Cefaclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one

Some ampicillin-resistant strains of *Haemophilus influenzae*—a recognized complication of bacterial bronchitis\*—are sensitive to treatment with Cefaclor.<sup>1-5</sup>

In clinical trials, patients with bacterial bronchitis due to susceptible strains of *Streptococcus pneumoniae*, *H. influenzae*, *S. pyogenes* (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Cefaclor.<sup>7</sup>

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hour. The effect on nursing infants is not known. Caution should be exercised when Cefaclor\* (cefaclor, Lilly) is administered to a nursing woman.

**Usage in Children**—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

**Adverse Reactions:** Adverse effects considered related to therapy with Cefaclor are uncommon and are listed below.

**Gastrointestinal symptoms** occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

**Hypersensitivity reactions** have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthritis and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during, or following a second course of therapy with Cefaclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome. Cases of anaphylaxis have been reported; half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

**Causal Relationship Uncertain**—Transient abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

**Hepatic**—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

**Hematopoietic**—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

**Renal**—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

(061782R)

\*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.

Note: Cefaclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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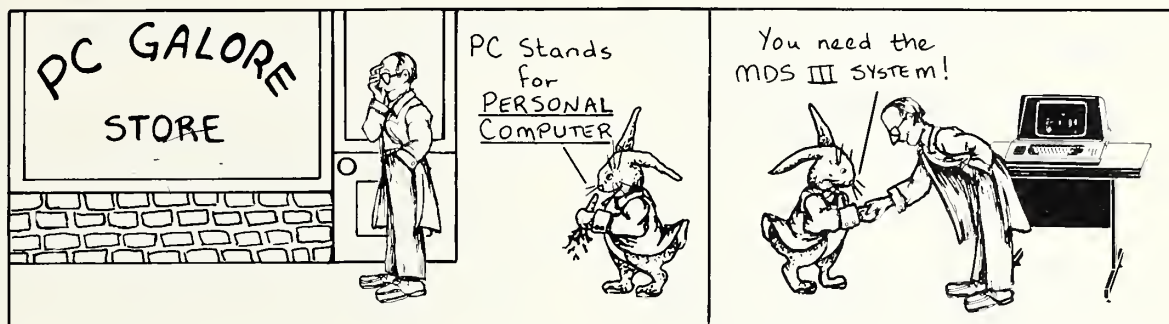


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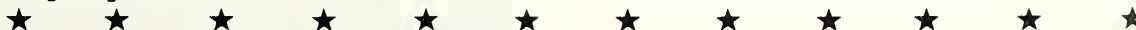
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## Head Injuries, Part II: Lessons from a Ten-Year Study

Susan E. Hellams, MD, and Donald P. Becker, MD,  
*Richmond, Virginia*

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Analyzing ten years of experience in treating head-injured patients at the Medical College of Virginia, the authors develop an optimal management program for brain trauma and document their recommendations with a review of the clinical and laboratory data.

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**O**VER the last decade, the outlook for the head-injured patient has been significantly improved, largely due to the research effort that has been made in understanding brain trauma. The Medical College of Virginia has been actively involved in this effort since 1973, when it became apparent that little was known about brain injury and that it represented a major medical problem, with over 700 Virginians dying from head injuries in 1973. Recognition of this problem led the MCV Neurosurgery Division to devote its efforts and resources to the study of head trauma. The initial hypothesis to be tested was whether aggressive

management and early diagnosis would improve outcome.

The primary group of patients investigated were those with severe head injuries. This was defined as anyone who, following resuscitation, was unable to follow commands. The initial hypothesis tested was "Can outcome from head injury be improved by more aggressive care?" A corollary to this question was the idea that perhaps following injury, groups of neurons were rendered temporarily dysfunctional but, given the proper internal milieu, could recover. With these goals, three tenets became the mainstay of care of the head-injured patient: 1) rapid triage; 2) early diagnosis and evacuation of mass lesions; and 3) intensive medical management to include controlled mechanical ventilation and monitoring of intracranial pressure (ICP). After analysing the results of 100 patients managed under these guidelines, we found that 57% made a good, functional recovery, 8% made a moderate recovery, 2% remained vegetative, and 32% died, a reduction in mortality of 40%.<sup>1</sup>

From the Division of Neurological Surgery, Department of Surgery, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Hellams at Box 631, MCV Station, Richmond VA 23298.

This is the second in a two-part series. Part I, "Solving Pre-Hospital Care Problems", appeared in VIRGINIA MEDICAL's February 1984 issue.

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These patients were then analyzed in a retrospective manner to determine what events surrounding their injuries may have affected their outcomes. It was found that a high percentage of the head-injured patients were hypoxic by the time of their arrival in an emergency room.<sup>2</sup> Several other factors were identified as detrimental to outcome, especially in the patient with a mass lesion.<sup>2</sup> These were: 1) hypotension, 2) anemia, and 3) hypercarbia.

Due to this information, MCV strengthened its advocacy of rapid transport of the head-injured patient to a major trauma facility without stopping at another hospital en route. This view was even more strongly supported by a study done in 1981 on patients with acute subdural hematomas. In this group of patients, the time from injury to the time of surgery was the single most important factor influencing outcome. In patients operated on within four hours after injury, a 30% mortality rate was seen, as compared to a 90% mortality rate seen in those patients operated on more than four hours after injury.<sup>3</sup>

Factors were also identified which aided in predicting outcome. These were 1) presence of a mass lesion; 2) age; 3) abnormal motor response; 4) impaired or absent eye movements or pupillary light response; 5) early presence of a systemic secondary insult; and 6) elevation of intracranial pressure over 20 mmHg (Table 1).<sup>4</sup> Of these, the presence of an intracranial mass lesion was the most important in affecting outcome.

In caring for the brain-injured patient, it was felt early on that monitoring the intracranial pressure was critical for three reasons: 1) as an early warning for the development of mass lesions; 2) as a guideline for the efficacy of therapy directed at lowering intracranial pressure, such as hyperventilation or mannitol; and 3) as a possible prognostic value. With these goals in mind, a device was developed at MCV to measure intracranial pressure commonly called the "Richmond bolt". This monitor, along with intraventricular catheters, has been employed at MCV for the last ten years. After monitoring a large number of patients, several facts became evident: 1) the ICP is almost always elevated in the patient with a mass lesion; 2) elevated ICP is associated with a poor outcome; specifically, in patients with mass lesions an ICP greater than 40 mmHg is associated with a poor outcome.<sup>6</sup>

Although a truly randomized trial has never been done to demonstrate its effect on outcome, intracranial pressure monitoring has continued to be employed as a standard procedure in the care of the

**Table 1. Factors Indicating Adverse Outcome Following Head Injury.**

Presence of a mass lesion
Advanced age
Abnormal motor response
Impaired or absent eye movements or pupillary light response
Early Presence of a systemic secondary insult, i.e., hypoxia, hypercarbia, or hypotension
Elevation of intracranial pressure over 20 mmHg

head-injured patient at MCV. However, there are risks associated with the use of an intracranial pressure monitor. These include infection and hemorrhage, with an overall complication rate at MCV of 5%-8%. We have felt that the therapeutic advantage of ICP monitoring far outweighs this low complication rate, especially since no mortality can be directly ascribed to this procedure. However, in an attempt to use ICP monitoring as judiciously as possible, a large group of patients were examined retrospectively in order to identify which patient population was most at risk for developing problems of elevated ICP and thus most likely to benefit from ICP monitoring. Using this approach, a high-risk group was identified which consisted of patients with any abnormality on CT scan. Patients with a normal CT scan were found to have a low risk of developing elevated ICP unless 1) they were over 40 years of age; 2) there was systolic hypotension under 90 mmHg; or 3) unilateral or bilateral motor posturing was present. If any two of these adverse features occurred, there was a 60% risk of intracranial hypertension, whereas if only one or none of these features existed, the risk was 4%. Using these criteria, approximately 60% of the severely head-injured patients would require intracranial pressure monitoring<sup>7</sup> (Table 2).

Other modalities have been used to follow the neurologic recovery of the head-injured patient. Of these, the most important has been multimodality evoked potentials. These include evaluation of the somatosensory, visual and auditory pathways from

**Table 2. Guidelines for Monitoring Intracranial Pressure.**

Monitor if there is
an abnormality on CT scan;
age over 40;
motor posturing (either unilateral or bilateral);
systolic hypotension >90 mmHg;
change in neurological exam despite normal CT scan.

**Table 3. Lessons Learned from the Care of Head-Injured Patients.**

- **Transport:** The head-injured patient should be taken to a major trauma facility. Stops en route at secondary hospitals should be avoided.
- **Airway:** Many head-injured patients are hypoxic following trauma. An airway must be maintained and ventilation assisted as necessary.
- **Resuscitation:** All external bleeding points must be controlled and fluids should be given liberally. Remember that the neurologic exam in the face of hypotension is meaningless.
- **Neurologic exam:** This should be performed early in the patient's course to establish a baseline and follow any change in the patient's condition.
- **Cervical spine:** Maintain in a neutral position until cleared by X-ray. Five percent of the head-injury population have an associated cervical spine injury.
- **Diagnosis:** A CT scan should be obtained as rapidly as possible.
- **Mass lesions:** Intracranial lesions should be surgically evacuated as soon as possible. A delay of longer than four hours can adversely affect outcome.
- **Prevention of secondary insults:** Hypotension, hypoxia, hypercarbia and anemia have been identified as having deleterious effects on cerebral function. Judicious care must be taken to avoid these insults, as they also have an adverse effect on patient outcome.
- **Intracranial pressure:** See Table 2 for monitoring guidelines. The intracranial pressure should be kept below 25 mmHg.
- **Medical care:** These patients are subject to a large number of complications, such as infection and fluid and electrolyte imbalance. The physician must be alert to these possibilities.

an electrophysiological standpoint and have been used routinely at MCV as another means of assessing neurologic dysfunction. Evoked potentials have proven to be good prognostic indicators, especially in those patients with medical complications during their recovery phase. In a large group of patients assessed in this manner, it was found that those with normal or mildly abnormal evoked potentials who had a secondary insult (such as hypoxia), with no change in the evoked potentials during this stress, continued to have favorable outcomes. However, if a deterioration in the evoked potentials occurred during a secondary insult, the patient was more likely to have a poor outcome. In this way, evoked potentials have been a dynamic means to follow the neurological function of patients with severe head injuries.<sup>8</sup> Multimodality-evoked potentials have also been used to correlate the patient's neurologic condition with the loci of trauma. Although previous work in animals using surgical lesions had implicated midbrain and brain-stem

injuries as correlated with motor posturing, early work with evoked potentials at MCV showed that the presence of decortication or decerebration depended on cerebral hemispheric dysfunction and not brain-stem dysfunction.<sup>9</sup> Abnormalities in oculocephalic responses and pupillary light responses were correlated with brain-stem dysfunction as evaluated by evoked potentials.

Based on a combination of clinical experience, retrospective analysis, and research findings, several lessons have been learned from the care of head-injured patients. These are outlined in Table 3 and represent basic guidelines which should be implemented by anyone involved in the care of a brain-injured patient. Within this framework, the following is a step-by-step patient management program based on the treatment protocol in use at MCV.

#### **At the Accident**

The patient should be evacuated promptly and the cervical spine maintained in a neutral position. Approximately 5% of patients with severe head injury will also have cervical spine injury. Many head-injured patients are hypoxic or apneic at the scene, with respirations weakened or agonal. The patient's mouth should be cleared of all debris, and assisted ventilation given. This may be given initially as mouth-to-mouth assistance or by inserting an oral airway and using an Ambu bag. If possible, an esophageal airway or an endotracheal tube should be inserted. One hundred percent oxygen should be used. If the patient is hypotensive, a survey for external bleeding sites must be made and a pressure dressing applied as indicated. If the patient's systolic blood pressure is below 70 mmHg and medical antishock trousers (MAST) are indicated, they should be used even in the presence of severe head injury to maintain cerebral perfusion.

A neurologic examination must be done at the accident scene. The most consistent manner to evaluate and transmit this information is based on the Glasgow Coma Scale. This evaluates three major areas: eye opening, motor response, and verbal response (Table 4). Pupillary size and light response should also be noted. It is crucial that a reliable and accurate baseline exam is obtained in order to follow the patient's course. A thorough survey of other body systems should also be made at the scene.

Transport the patient in the supine, horizontal position with the cervical spine fully stabilized. This makes the patient fully accessible. Vital signs and the neurological exam should be monitored every five minutes. One hundred percent oxygen should



be administered through an endotracheal or esophageal airway. If no airway has been inserted, nasal oxygen should be given at 3-5 L/min. Lactated ringers or other saline solution should be given during transport at 100-150 cc/hr in the adult. This should be administered through a large-bore intravenous catheter, preferably 16 gauge. Fluid restriction should not be used in the presence of a head injury.

Radio contact should be established with the receiving hospital as soon as possible and the neurosurgeon advised of the patient's condition. If further deterioration in the patient's condition should occur, such as the onset of posturing or the development of a unilaterally dilated pupil, the ambulance squad under the direction of a physician may administer an osmotic agent, such as mannitol one gm/kg IV. This should be done only under the direction of a physician as hypotension may develop when the diuresis begins. Given in the right circumstances, its administration may be lifesaving.

#### In the Emergency Room

Based on the experience at MCV, we recommend transport of the patient to a full-scale trauma center where a neurosurgeon, general surgeon and other staff members are available 24 hours a day. On arrival at the emergency room, priority should be placed on fully stabilizing the patient and preventing such secondary insults as hypoxia and hypotension. Because multiple injuries are common in the severely head-injured patient, the resuscitative team should include a general surgeon, orthopedic surgeon, anesthesiologist and neurosurgeon. All injuries need to be appraised during this time and appropriate diagnostic procedures planned. It is critical to clear the cervical spine of injury and this

is best done by obtaining a lateral and AP view of the spine through the C7-T1 interspace. Once the cervical spine has been cleared of injury, intubation, if not already performed, will be much easier. A definitive airway should be placed, preferably an endotracheal tube. If multiple facial injuries are present with massive soft tissue swelling, or oral or pharyngeal injuries exist, intubation by this route may be impossible. In this case, no hesitation should exist in performing a tracheostomy or cricothyroidotomy in the emergency room. Following intubation, the patient should be artificially ventilated with a tidal volume of 15 ml/kg. The pCO<sub>2</sub> should range between 25-28 and 100% oxygen should be used initially.

The patient should be immediately evaluated by a neurosurgeon with particular attention placed on verbal response, ability to follow commands, motor exam, pupillary size and light response. If no cervical spine injury is present, the oculovestibular response should be noted. If any unilateral findings are present with evidence of incipient herniation, mannitol one gm/kg should be given as an IV bolus. Other signs of neurologic injury should also be looked for, including hemotympanum, echymosis over the mastoid process, or periorbital echymosis as evidence of a basilar skull fracture. Any CSF leak should be noted.

A careful general physical exam must also be done, with the patient evaluated for evidence of chest trauma, facial fractures, orbital injuries, blunt abdominal trauma, and fractures of the long bones and pelvis. All of these are common injuries in the head-injured patient.

The patient should be hemodynamically stabilized while in the emergency room. The blood pressure should be maintained by the use of crystalloids and blood products as necessary. If hypovolemic shock is present, the cause must be established, because head injury alone never causes hypovolemic shock. Also, a neurologic exam in the face of hypotension is meaningless.

In addition to examining the patient, laboratory studies should be ordered, including hemoglobin, hematocrit, white blood cell count, blood chemistries and blood alcohol level. Arterial blood gases should be ordered. A Foley catheter should be inserted for an accurate assessment of the patient's fluid status and as a check for urological injuries. A diagnostic peritoneal lavage is recommended in all comatose patients in whom blunt abdominal trauma is suspected. If positive, an exploratory laparotomy will need to be performed for possible splenic rupture or hepatic laceration.

Table 4. Glasgow Coma Score.

Eye opening	spontaneous	4
	to sound	3
	to pain	2
	none	1
Motor response	obeys commands	6
	localizes pain	5
	normal flexion	4
	abnormal flexion (decorticate)	3
	extension (decerebrate)	2
	none	1
Verbal response	oriented	5
	confused conversation	4
	inappropriate words	3
	incomprehensible sounds	2
	none	1

An X-ray survey should be done to include skull films, facial films, chest, abdomen, pelvis, lateral thoracic and lumbar spine, and any long bones that are indicated by clinical exam. This series of films can be obtained in 15 minutes with an efficient group of X-ray technicians and a portable machine.

The head-injured patient should also receive anti-convulsant prophylaxis against seizures while in the emergency room. We currently give phenytoin sodium (Dilantin®) 250 mg IV over a five-minute period and phenobarbital 60 mg IV. Currently, steroids, such as dexamethasone (Decadron®) are not being used at MCV. Their use in head injury remains an unsolved issue, although they seem to offer little in traumatic brain injury. Tetanus prophylaxis should also be given.

A CT scan should be obtained once the emergency room evaluation is complete and the patient stable. It is critical that a high-quality scan without motion artifact be obtained. This is best accomplished in the intubated patient by giving pancuronium bromide (Pavulon®) 2-4 mg IV in order to temporarily paralyze the patient. If a major shift with a mass lesion is seen, the patient should be immediately taken to the operating room (Fig. 1). Mass lesions requiring surgery are generally considered to be those which cause a shift of 5 mm or more across the midline and may be due to a subdural hematoma, epidural hematoma, intracere-

bral hematoma or contusion. If the shift is less than 5 mm, a decision to operate is made based on the patient's condition (Fig. 2).

Following surgery or the CT scan, the patient should be returned to an intensive care unit where controlled artificial ventilation is continued. A decision to monitor the intracranial pressure should be made based on the criteria discussed earlier. Those patients with mass lesions and those who are over 40 years of age and are hypotensive with a systolic blood pressure less than 90 mmHg and those exhibiting motor posturing should be monitored, as they stand a high risk of developing intracranial hypertension. The decision to monitor a patient with a normal CT scan is based on clinical exam and any deterioration which might occur. A ventricular catheter or subarachnoid bolt may be used, with the catheter offering the advantage of CSF drainage should elevated intracranial pressure occur. Any ICP over 25 mmHg should be considered elevated and treated. This can be done by several methods, including hyperventilation or drainage of CSF if an intraventricular catheter is present. If these two methods are ineffective, a bolus of mannitol one gm/kg IV may be used. If mannitol is used, the patient's fluid balance and electrolyte status must be carefully followed. Frequently, patient agitation may lead to ICP elevations, and in this case morphine 2-4 mg IV may be helpful. If all these methods

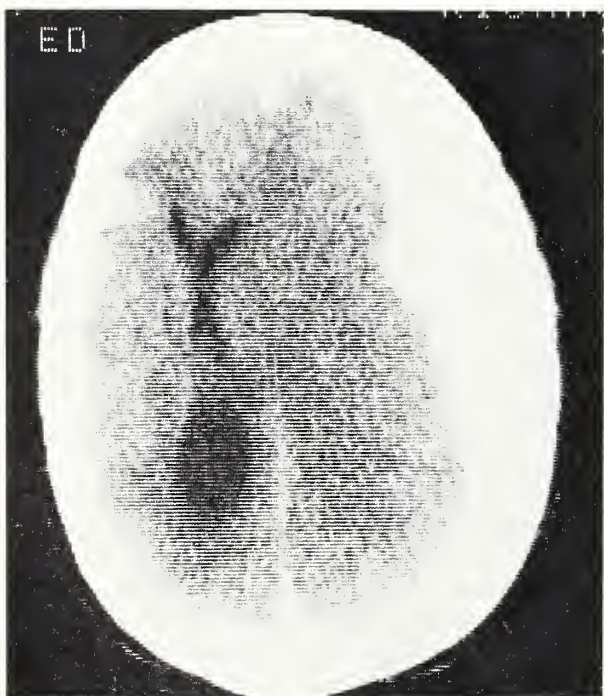


Fig. 1. On this CT scan, large right subdural hematoma with 9-mm shift of ventricular system indicates the need for immediate surgical decompression.

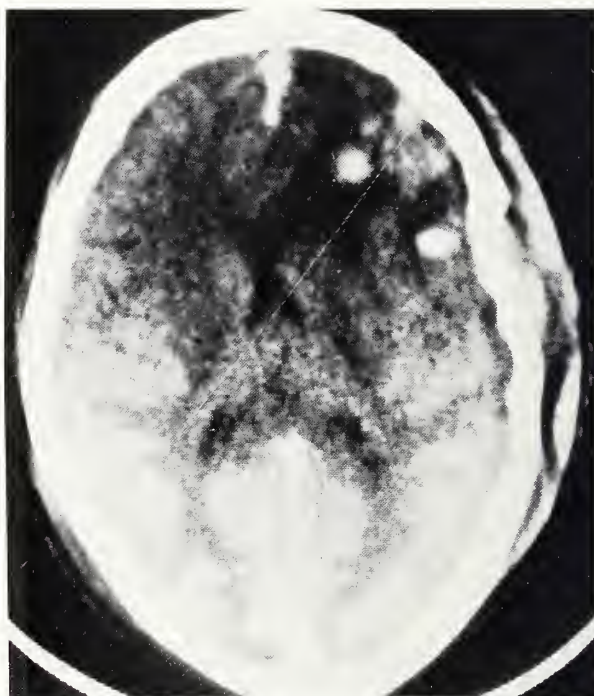


Fig. 2. This CT scan shows right frontal contusion with only slight shift of ventricular system, indicating patient can be managed medically.



are ineffective, a bolus of pentobarbital 5-10 mg/kg IV may be tried.

Any unexplained rises in ICP must be fully investigated and the patient evaluated for hypoxia, hypotension, sepsis or hyponatremia. Also, the patient's position in bed should be noted, as obstruction of venous return may elevate ICP. If no underlying cause for an ICP rise has been found at this point, a repeat CT scan should be done to rule out a delayed bleed, increase in size of an intracerebral hematoma, or the presence of hydrocephalus. If none of these are found, the elevated ICP can be attributed to an increase in cerebral edema or cerebral blood volume. This stage requires a combination of therapeutic modalities until the crisis has passed.

A patient with a severe head injury should be maintained on Dilantin 100 mg IV Q8h, and phenobarbital 60 mg IV q8h (with adjustments as necessary to maintain therapeutic levels) and cimetidine 300 mg IV q8h. Repeat CT scans should be done at regular intervals. A constant guard must be maintained against the numerous complications these patients are prone to, including acute respiratory distress syndrome, pneumonia, disseminated intravascular coagulopathy, gastrointestinal bleeding, ventriculitis, meningitis, urinary tract infections, sepsis, diabetes insipidus, syndrome of inappropriate antidiuretic hormone secretion, as well as a variety of others. Nutritional support, either by tube feedings or hyperalimentation, should begin once the general condition of the patient has stabilized.

Ultimately, as the patient's intracranial process stabilizes and the intracranial pressure remains normal, ICP monitoring is no longer necessary. At this point, the patient can be gradually weaned from the respirator, oral feedings begun, and intensive work begun by speech pathologists, physical therapists and occupational therapists. During this rehabilitative phase, neuropsychological testing should be done to establish a baseline for the future. Anticonvulsants should be continued during this time and one drug continued for at least one year post-trauma regardless of the patient's seizure history.

This recovery period is a demanding time on both the patient and the family as each tries to accommodate to new physical and mental limitations on the part of the patient.

The rewards of such an intensive management program are reflected in our statistics on patient outcome. A 40% reduction in patient mortality coupled with an increase in those patients with a good, functional outcome has made working with head-injury patients a rewarding and gratifying experience at the Medical College of Virginia. This goal has been realized through a comprehensive management program which begins at the roadside and continues through the patient's entire post-trauma course.

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# Vascular Transposition for Vertebral Basilar Insufficiency

Ralph B. Pfeiffer, Jr., MD, Stanley O. Snyder, Jr., MD,  
Roger T. Gregory, MD, Robert G. Gayle, MD, and  
Jock R. Wheeler, MD, *Norfolk, Virginia*

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Surgery has not traditionally been the treatment of choice for symptoms of vertebral basilar insufficiency, but the authors performed vertebral to carotid artery transposition in 14 patients with these symptoms, finding severe stenosis in all. Nine patients were relieved of symptoms, four were improved, and one remained unchanged.

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**A**LTHOUGH it has been 25 years since the first surgical correction of a vertebral artery stenosis, little progress has been made in the evaluation, patient selection and general acceptance of this aspect of vascular surgery.<sup>1</sup> This retrospective study outlines the results in 14 patients operated on for vertebral artery stenosis over a two-year period, representing approximately 4% of the total cerebrovascular surgical procedures performed over the same period.

## Materials and Methods

All 14 patients presented with typical vertebral basilar transient ischemic symptoms. This included

incapacitating dizziness, ataxia, diplopia, blurred vision, paresthesias and loss of consciousness. There were nine men and five women, ranging in ages from 47 to 78 (mean 65). EEG, CT brain scan, isotope brain scan, Holter monitor or neurological consultant were performed as the clinical situation warranted.

Arch angiography was performed on all patients to demonstrate the vertebral artery origins and extracranial carotid arteries. Selective carotid and vertebral studies were performed on six patients.

Patients were selected for surgery if there was significant vertebral artery disease and their preoperative assessment demonstrated no other definitive cause for vertebral basilar insufficiency. Hemodynamically significant carotid artery stenoses were corrected prior to vertebral artery surgery. Bilateral vertebral artery stenosis or unilateral stenosis with contralateral hypoplasia or occlusion was demonstrated in all patients. The vertebral artery stenosis was considered significant only if it was greater

From the Department of Surgery, Vascular Division, Eastern Virginia Medical School. Address correspondence to Dr. Snyder at 250 West Brambleton Avenue, Norfolk VA 23510.

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than 75%. No procedures were done in conjunction with other vascular procedures and none were performed for subclavian steal. Angiograms or Sonacolor 6000® were used postoperatively to assess patency of the transposed vertebral artery.

### **Surgical Technique**

A seven- to eight-centimeter supraclavicular incision is made from the suprasternal notch extending laterally, as outlined by Edwards and Mulherin.<sup>2</sup> The sternocleidomastoid, sternohyoid and sternothyroid muscles are transected to expose the common carotid and internal jugular vein. The carotid artery and jugular vein are then mobilized for several centimeters longitudinally to facilitate dissection between the two. The vagus nerve is identified and carefully preserved by retracting it laterally with the jugular vein.

Exposure of the scalene anticus muscle and phrenic nerve is not necessary. The thoracic duct is encountered when operating on the left vertebral and should be carefully ligated. The pre-vertebral fascia is then incised, exposing the vertebral vein, which must be carefully ligated. Small sympathetic ganglia and nerve roots are frequently found anterior to the vertebral artery and may need to be transected in order to expose sufficient length of artery. However, preservation of these structures will prevent development of Horner's syndrome.

The proximal two to three centimeters of vertebral artery are exposed from its subclavian origin. The common carotid artery should also be mobilized for an arteriotomy. The transposition is performed by transecting the vertebral artery and anastomosing it end-to-side to the posterior, lateral aspect of the common carotid artery, utilizing continuous six-0 polypropylene suture. No shunt is used in the common carotid artery and the wounds are closed in layers without a drain. The entire operation is performed using 2.5 power magnification. Vertebral artery flows are obtained before and after the transposition when technically feasible.

### **Results**

Nine patients were completely relieved of all of their symptoms prior to discharge from the hospital and have remained so during the study. Four patients had marked symptomatic improvement of at least one of their symptoms, and one was unchanged.

Of the four patients who were not totally relieved of their symptoms, three had complete relief of at least one complaint. One patient suffered a postoperative stroke on the contralateral hemisphere and

could not be adequately evaluated for symptomatic improvement. An immediate postoperative angiogram in this patient demonstrated patency of the vertebral-to-carotid anastomosis as well as widely patent carotid arteries bilaterally; one week prior to the vertebral procedure this patient had developed hemispheric transient ischemic attacks and had undergone a left carotid endarterectomy for an ulcerated plaque.

The 14 patients were followed an average of 15 months, ranging from five months to 25 months. All have been followed in the office for the duration of the study.

Four patients had postoperative angiograms to demonstrate patency of the anastomoses. Nine patients were evaluated in the non-invasive laboratory with Sonacolor 6000 and Dopscan®. They were found to have antegrade vertebral flow on the operated side. One patient expired at 18 months from a GI bleed and could not be reassessed, and one patient would not cooperate with the followup evaluation. One Dopscan examination was technically unsatisfactory for adequate interpretation.

Five patients developed transient Horner's syndrome, which gradually cleared over two to 20 weeks. Since sympathetic ganglia and nerve roots lie anterior and adjacent to the vertebral arteries, this postoperative complication is not unusual. No patient developed a lymphocele, recurrent laryngeal nerve injury or other complications, and there were no postoperative deaths.

Vertebral artery flow measurements obtained before and after transposition were recorded on three patients. Technical problems relating to the size and shape of the flow probes prevented complete evaluation in most patients. Of those tested, the flows increased from an average of 100 cc/min to 159 cc/min following transposition.

### **Discussion**

Since vertebrobasilar transient ischemia is a syndrome difficult to define, it is understandable that surgical correction for this symptom complex by vertebral artery revascularization is controversial. Few large series other than Edward's have been reported that demonstrate the efficacy of vertebral revascularization.<sup>3</sup> He achieved an 85% success rate using a similar surgical approach. Buerger and Bauer recently reported a 90% success rate in 14 patients utilizing a vein graft between the vertebral artery and the subclavian arteries to bypass the vertebral stenosis.<sup>3</sup> Imparato, et al also recently reported favorable results on 58 patients who underwent vertebral angioplasty for severe stenosis.<sup>4</sup>

Other scattered reports mention successful vertebral artery surgery using a variety of techniques.<sup>5-7</sup>

Direct vertebral-to-common-carotid-artery anastomosis has several advantages over other techniques. The operation can be performed through a single, supraclavicular incision, requiring only one anastomosis. Dissection of the subclavian artery is kept to a minimum and no graft material is required. Brief occlusion of the ipsilateral carotid artery for the anastomosis is a theoretical concern, but it has not proven to be a significant problem, as the level of carotid occlusion allows the external carotid artery to function as a potential collateral to the internal carotid.<sup>2</sup> It has been previously documented that the common carotid artery can be used as an inflow vessel without arterial steal when there is no significant carotid artery stenosis.<sup>8</sup>

Powers, Narittoni and Khodadad describe dizziness, diplopia, ataxia, paresthesias and syncope as symptoms of hypoperfusion of the basilar artery and its branches.<sup>9-11</sup> These symptoms may have multiple etiologies. Careful exclusion of cardiac abnormalities, brain pathology, ENT problems and a variety of neurological disorders is important to properly select patients with specific vertebrobasilar symptoms who might benefit from revascularization.

Our patients were selected for vertebral artery transposition when medical, neurological and cardiac evaluation revealed no other obvious source of symptoms. In these patients, angiography demonstrated a stenotic vertebral artery in conjunction with a hypoplastic stenotic or occluded vertebral on the contralateral side and no significant carotid artery stenosis. With this method of selection, over 90% of this small group of patients were either relieved of symptoms or significantly improved following surgery.

Vertebral basilar symptoms have been treated by carotid endarterectomy by Rosenthal and others. He reported favorable results following carotid endarterectomy for vertebral basilar symptoms, but it is interesting to note that only 20% of his patients had vertebral basilar symptoms alone.<sup>12</sup> McNamara, on the other hand, has reported poor results following treatment of vertebral basilar insufficiency with carotid endarterectomy.<sup>13</sup> The carotid's contribution to the posterior circulation may be over-emphasized since more than 50% of humans have some abnormality in the Circle of Willis, and 25% have anomalous posterior communicating arteries.<sup>14</sup> Riggs and Rupp also found hypoplasia of one or both components of the Circle of Willis in 79% of 994 patients who had evidence of brain

pathology in autopsy specimens.<sup>15</sup>

Based on our experiences in this retrospective study, a more aggressive evaluation of vertebral basilar insufficiency symptoms is recommended. Ideally, patient evaluation should include an EEG, Holter monitor, CT brain scan, arch and selective carotid and vertebral and intracranial angiography, and neurological and ENT evaluations. Properly selected symptomatic patients with vertebral artery stenosis can benefit from vertebral artery transposition.

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# Virginia's Hidden Enemy



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# Blunt Esophagectomy: a Misnomer

Irving L. Kron, MD, Axel Joob, MD,  
M. E. Johns, MD, and G. R. Minor, MD,  
*Charlottesville, Virginia*

Over a 14-month period, esophagectomy without thoracotomy proved a valuable adjunct in 12 selected patients with carcinoma of the esophagus or hypopharynx. There were no complications involving bleeding or anastomotic leaks. The death of a 78-year-old patient at 30 days was related to chronic aspiration.

**B**LUNT esophagectomy was first described in 1913.<sup>1</sup> The technique was repopularized in 1966<sup>2</sup> for use in the replacement of the cervical esophagus and then, in 1978,<sup>3</sup> for all levels of esophageal carcinoma.

The technique features an esophagectomy done without a thoracotomy, using blunt dissection in the posterior mediastinum through the esophageal hiatus from the abdominal approach and from a neck incision. Actually, the procedure is misnamed. The only real blunt mobilization is performed at the level of the carina, which is an area of minimal

From the Department of Surgery (Drs. Kron, Joob and Minor) and the Department of Otolaryngology/Head and Neck Surgery (Dr. Johns), University of Virginia. Address correspondence to Dr. Kron at Box 181, University of Virginia Medical Center, Charlottesville VA 22908.

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blood supply to the esophagus, so that most blood vessels can be visualized and clipped.

There was a great deal of controversy regarding the procedure's indication and usage when it was more recently presented. Ronald Belsey, the noted British esophageal surgeon, termed it an "expedition into the dark ages."<sup>4</sup> Nevertheless, certain advantages seem obvious. Blunt esophagectomy avoids thoracotomy in those patients who might not be good candidates for esophageal resection through that procedure. As a technique, it provides a method of bringing the stomach to the neck to replace the cervical esophagus and to avoid the use of skin flaps as a means of replacing the cervical esophagus for complicated head and neck malignancies.

Because of these advantages, we decided to perform these procedures at the University of Virginia for certain selected patients, including those patients who required the stomach as a means of replacing the cervical esophagus or hypopharynx in selected patients with head and neck malignancy. It was also used in patients with carcinoma of the upper thoracic or mid-thoracic esophagus who were felt to be unsuited for thoracotomy.

## Patients

From March 1982 to May 1983, 12 patients with carcinoma of the esophagus or hypopharynx were entered into the series. The age range was 48-78 years, with an average age of 63. All 12 patients had squamous carcinoma; there were four tumors of the hypopharynx, four tumors of the cervical esophagus, and four of the mid-thoracic to upper thoracic esophagus. Seven patients had had one to four previous head and neck malignancies, which had been either resected or irradiated.

## Technique of Operation

A two-team approach is used. The abdominal portion is done through a vertical upper-midline abdominal incision. Either a collar-neck incision is used in patients who are to have pharyngolaryngectomy, or a linear incision is made along the anterior border of either sternocleidomastoid muscle. The stomach is mobilized, preserving the right gastric and right gastroepiploic arteries. A pyloroplasty is performed in all cases. The esophageal hiatus is manually widened while a Penrose drain is placed around the esophagus at the hiatus. The esophagus can be mobilized; all vessels can be visualized up to the level of the carina and secured with clips through the hiatus (Fig. 1). From the cervical approach, after mobilizing the cervical esophagus, the



dissection is carried inferiorly beyond the azygous vein. Most major blood vessels can be visualized and clipped. The cervical esophagus is transected at the appropriate level above the tumor, and a Penrose drain is sutured to the divided lower end. The latter is then brought down through the hiatus with the Penrose drain attached, and the esophagus is transected at the level of the gastroesophageal junction with a TA-55 stapler. The Penrose drain which is now lying in the posterior mediastinum is attached to the gastric fundus with a suture, and is then used to bring the gastric fundus to the level of the cervical esophagus. A two-layer standard anastomosis is then performed, and a drain is left in place through the neck incision. Inspection for bleeding and standard closure of the abdominal incision are performed.

### Results

In two of the patients who had resection for cervical esophageal carcinoma, a second, previously unknown tumor was noted in the esophagectomy specimen. Preoperative esophagoscopy and barium swallow had not revealed these second tumors.

The estimated blood loss was 500 cc when no laryngopharyngectomy was performed and approximately 1000 cc with laryngopharyngectomy. There were no complications involving bleeding or anastomotic leaks. Three patients who had had previous cervical esophageal radiation developed strictures which could be dilated with Maloney dilators. There was one death in the series, a patient who had a tumor that reached the level of the nasopharynx. He could not undergo primary reconstruction with stomach, and as a result, had a cervical gastrostomy placed. Unfortunately, he developed chronic aspiration into his tracheal stoma and finally died of respiratory failure. He was also the oldest patient in the series, at age 78.

The average operative time when no pharyngeal laryngectomy was performed was between two and three hours. The average hospital stay for those patients who had a primary gastrointestinal reconstruction was ten days.

### Comments

We have found that an esophagectomy without thoracotomy has been very helpful in certain selected cases. We have used it to help us handle difficult patients with recurrent hypopharyngeal tumors following laryngectomy. It has also been helpful in those patients with mid-thoracic or upper-thoracic esophageal lesions with reduced pulmonary or cardiac reserve. The operation has turned out to be a

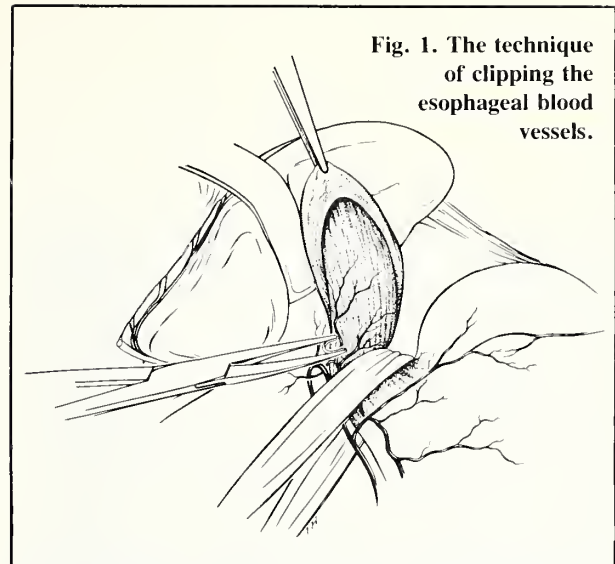


Fig. 1. The technique of clipping the esophageal blood vessels.

safe operation, and almost all the major blood supply to the esophagus can be visualized and occluded with steel clips. Bleeding has not been a problem for our patients. We have used computerized axial tomography to determine those tumors that are appropriate for transabdominal esophagectomy.<sup>5</sup> Those patients whose tumors have been shown to be confined to the esophagus itself without direct extension into vital structures have been felt to be candidates for esophagectomy without thoracotomy. However, those patients who have been shown to have extension of the tumor beyond the esophagus have undergone standard esophagectomy, and in all cases could not have been treated safely by the so-called "blunt esophagectomy."

We believe this technique has served and will continue to serve as a valuable adjunct in the treatment of patients with carcinoma of the esophagus or hypopharynx, in that it avoids thoracotomy in selected patients and also allows removal of the entire esophagus, which in some of these patients may harbor unknown second primary tumors.

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# Prenatal Sonographic Diagnosis of Osteogenesis Imperfecta: Case Reports

Rolfe D. White, MD, *Virginia Beach*;  
Paul E. Lewis, MD, *Portsmouth, Virginia*;  
and Roger C. Sanders, MD,  
*Baltimore, Maryland*

**O**STEOGENESIS imperfecta, also known as osteopsathyrosis idiopathica, Lobstein disease, Eddowes's syndrome, fragilitas ossium, Vrolik disease, and Spurway disease, is a rare, inherited multi-system disease affecting the mesenchyme and its derivatives. It results most often in three clinical entities—bone fragility, blue sclera and otosclerosis. Less frequently, skull deformities, prognathic chin, exophthalmos and dentinogenesis imperfecta are seen. Numerous endocrine, hematologic and metabolic disorders may be associated with this condition.

These two cases demonstrate that osteogenesis imperfecta can be diagnosed if the sonographic and roentgenographic examinations are compared.

## Case Report No. 1

A 19-year-old patient, married, white, gravida 2,

From the Departments of Obstetrics and Gynecology, Eastern Virginia Medical School (Dr. White) and Naval Regional Medical Center (Dr. Lewis), and the Department of Radiology, Johns Hopkins University (Dr. Sanders). Address correspondence to Dr. White at 3386 Holland Road, Virginia Beach VA 23452.

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para 0, abortus 1, initially seen at 17 weeks by dates and by size, stated that she and her husband had an unremarkable family and past medical history. Her prenatal course was uncomplicated, until at 33 weeks by size and by dates she presented to the obstetric admission room with a history of spontaneous rupture of membranes. This was confirmed by speculum examination, which revealed gross amniotic fluid in the vault. The fluid was positive for ferning and had an alkaline pH. On abdominal examination, the fetal head was not palpable. A pelvic examination later revealed a 3-cm dilated cervix and a high abnormal presenting part consisting of soft and bony areas, not clearly identifiable. A standard fetogram revealed faintly visible vertebrae and an absent skull (Fig. 1). As anencephaly or poor radiographic technique was suspected, a gray scale ultrasound examination was then performed (Fig. 2). Although initially read as normal, it was later noted that the cerebral gyri were abnormally clear, the skull tables extremely thin, and the head markedly wedge-shaped for such high station in early labor. After contractions of approximately four hours, the fetus was noted to have absent fetal heart rate variability and severe variable and late decelerations. Cervical dilatation was 4 cm. A primary cesarean section for fetal distress was then performed, delivering a 3 lb 10½ oz female infant with Apgar scores of 6 and 7 at one and 5 minutes, respectively, with an extremely soft skull and short fractured limbs. The infant died approximately 36 hours after birth. The histologic and gross findings at the postmortem examination confirmed the diagnosis of osteogenesis imperfecta (Type II). In addition, a parieto-occipital subarachnoid hemorrhage was noted.

## Case Report No. 2

A 24-year-old patient, married, white gravida 1, para 0, abortus 0, was noted at approximately 28 weeks gestation to have polyhydramnios and edema. Fetal heart tones could not be auscultated. A fetogram was obtained, revealing poor calcification with no evidence of limbs or cranium. The presence of abnormal vertebral bodies was also noted.<sup>1</sup>

Family, past medical, prenatal and environmental histories were all non-contributory except for a history of spontaneous rupture of membranes several hours prior to the examination. Fundal height was approximately 30 cm. Ruptured membranes were confirmed by demonstrating ferning of the fluid on microscopic examination. Fetal heart tones were identified by doppler.

As an abnormal presenting part was appreciated



on pelvic examination and a fetal skull was not appreciated on fetogram, anencephaly was suspected. Bistable sonography, however, revealed a single vertex fetus with a biparietal diameter of 8.4 cm. Given this information, the sonographer suspected the diagnosis of osteogenesis imperfecta.

Approximately 28 hours later, a 30-to-32-week dismembered, decapitated, stillborn fetus was delivered vaginally. The postmortem examination, gross and histologic findings, were consistent with the pathological diagnosis of osteogenesis imperfecta (Type II).

### Discussion

Most authors agree that the incidence of osteogenesis imperfecta is between 1:20,000 and 1:60,000 deliveries. There is considerable variation in the clinical presentation of this disease. The most frequently used classification divides this spectrum into four types. Type I (autosomal dominant) patients have blue sclerae and less severe skeletal deformities. Type II (autosomal recessive) patients have severe deformities usually incompatible with life. They have blue sclerae, marked lack of osseous mineralization, multiple fractures, limbs that are shortened, angulated and bowed, and may have collapsed crania. Type III (autosomal recessive) patients have less severe demineralization and deformities. They have normal sclerae and usually survive. The Type IV patients also have normal sclerae, even less deformities and generally will have a normal lifespan, although they frequently encounter long bone and rib fractures.<sup>2</sup>

Both osteogenesis imperfecta and hypophosphatasia may occur spontaneously. Hypophosphatasia is a disease with a similar presentation, usually considered in the differential diagnosis of excessive demineralized bone. The majority of these cases are autosomal recessive. Although over half of the patients with the early infant type of hypophosphatasia do not survive infancy, there is a wide spectrum of severity.

These two cases were similar in that breech presentations were initially suspected. Radiographs did not demonstrate a calcified fetal skull and were interpreted as indicating anencephaly; the skulls were visible, however, on sonograms. Further examination of the radiographs showed poor ossification of the bones and an absence of the petrous ridges normally visualized in anencephaly. Sonographic findings were abnormal in that the skull outline was very poorly defined, the normal ovoid shape of the fetal head was exaggerated, and the cerebral gyri and ventricles were abnormally clear

and well-defined. Rib and long-bone abnormalities were not noted.

Hypophosphatasia and osteogenesis imperfecta may be diagnosed with sonography, amniocentesis and radiography early enough in pregnancy for possible therapeutic intervention. However, as in these two cases, the possibility of abnormality for



Fig. 1. Standard fetogram shows absence of fetal calvarium cephalad to scalp electrode.



Fig. 2. Sagittal mid-line sonogram reveals abnormal skull shape and thin skull tables.

most patients may not present until the third trimester or even labor.

As has been suggested by Zenuoudakis et al, the fetal monitor tracing of a fetus with osteogenesis imperfecta in labor may demonstrate severe early decelerations because of the compression of the soft fetal vertex, variable decelerations because of the absence of the normal cord protecting bones, and late decelerations because of associated placental factors. Various patterns may be influenced as well by a congenitally abnormal fetal heart. Fetal distress and demise are not uncommon.<sup>3</sup>

Since these diseases present with a wide spectrum of severity and definite antenatal diagnosis may not be made, the advantage of abdominal versus vaginal delivery is yet to be seen. In those cases where meaningful survival is possible, cesarean section would seem preferable.

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The second edition of Virginia Medical's Directory of Medical Society of Virginia members will come to you in the August 1984 issue. Your name and address will appear as shown on your mailing label, which you will find on the back cover of this issue. Is it correct? If not, send your revisions to Virginia Medical, 4205 Dover Road, Richmond VA 23221.

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There are many excellent handbooks of effective writing, among them *The Elements of Style*, by William Strunk, Jr., and E. B. White (MacMillan); *The Careful Writer: A Modern Guide to English Usage*, by Theodore M. Bernstein (Atheneum); and *How to Write and Publish a Scientific Paper*, by Robert A. Day (ISI Press)



# Perinatal Diagnosis of Complete Congenital Heart Block: Four Cases

Raul A. Lazarte, MD,  
*Falls Church, Virginia,*  
and Edward Goldson, MD,  
*Denver, Colorado*

COMPLETE congenital heart block is encountered infrequently by the obstetrician. Its incidence has been reported at 1:20,000 births by Michaelsson and Engle, based on joint European-American study in 1972.<sup>1</sup> Roberts and Gelband,<sup>2</sup> however, estimated the incidence at 1:2,500 births and Esscher<sup>3</sup> feels this higher incidence to be more nearly correct.

In this paper we report our experience with four infants born with complete congenital heart block, all followed into late childhood.

## Subjects

The four children reported were born between 1970 and 1979 in the Denver metropolitan area and were transported to the Children's Hospital, where they received their initial medical care and were followed. Two are females and two are males. Three were full-term infants with birthweights ranging between 2892 and 3380 grams and had 5-minute Apgar scores of 9. One male was a 3340-gram, 33-week premature infant, severely hydropic at birth,

From the Department of Pediatrics/Neonatology, Fairfax Hospital, 3300 Gallows Road, Falls Church VA 22040, and Children's Hospital, Denver. Address correspondence to Dr. Lazarte.

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who had 1- and 5-minute Apgar scores of 4 and 2. He has previously been reported.<sup>4</sup>

## Medical History

In all four cases there was concern, prenatally, because of fetal bradycardia (heart rate below 100) or fetal heart tones that were difficult to detect by the obstetrician. Two of the children were delivered by cesarian section because of the fetal bradycardia. Following birth, three infants went into congestive heart failure within the first week of life. The electrocardiograms (EKG) on all of the children showed ventricular rates ranging from 40 to 60 beats per minute with evidence for complete heart block. All of the children had cardiomegaly demonstrated by radiographs of the chest.

For the three infants in overt heart failure, treatment with furosemide, isoproterenol and digoxin was unsuccessful, so temporary transvenous pacemakers were placed. Two of the children required temporary pacing within the first 48 hours of life and one within the first week. Permanent pacemakers were placed in one case in the first month of life, in two cases within the first year of life and in one case by three years.

Three of the children had other cardiac anomalies. One child had a corrected transposition of the great vessels with a coarctation of the aorta. The coarctation was repaired shortly after birth. Two children had atrial septal defects (ASD) of the secundum type which have been repaired. All of these children had the diagnosis of congenital heart block prior to the surgical correction of their cardiac anomalies. One child had no associated cardiac defect. None of the children have family histories of congenital heart block.<sup>5</sup>

Of note is the fact that the mother of the hydropic infant, who also had an ASD, was later found to have systemic lupus erythematosus, confirmed by ANA test.<sup>4</sup> The remaining mothers were symptom-free up to the last evaluation prior to this report, with ANAs reported negative. Repeat ANAs have been suggested to the physicians of these families. There are no histories of other collagen vascular disease.<sup>6</sup>

The oldest child has had two revisions of her pacemaker, had her ASD repaired at 9 years of age, and at 10 years of age developed hypothyroidism, which is now under control with medication. The second oldest child had *Hemophilus influenzae* meningitis, subdural effusions and a cardiorespiratory arrest at 6 months of age; he had a repair of his ASD at 4 years of age and two months after surgery required a pacemaker revision. The third oldest

Table 1. Cardiac Findings in Four Children with Congenital Heart Block.

Case/Sex	Obstetrical History	Presenting Signs	Atrial Rate	Ventricular Rate	Cardiac Defect
1 Female	Bradycardia, cesarian section	At 7 days of age heart rate 40/min and grade 1/6 systolic ejection murmur. At 6 wks congestive heart failure	170	50	ASD
2 Male	Bradycardia, emergency cesarian section	At birth hydrophic, in congestive heart failure, heart rate 40/min and grade 2/6 systolic ejection murmur	150	40	ASD
3 Male	Bradycardia	At 3 hrs of age heart rate 44/min and grade 3/6 systolic ejection murmur. At 17 days of age congestive heart failure	135	50	None
4 Male	Fetal heart tones difficult to detect	At birth cyanotic spells, heart rate 60/min and systolic ejection murmur	135	60	Coarct, CTGA

child had a pacemaker revision at 5 years of age, after we evaluated him. The youngest child has had no revisions to date (Tables 1 and 2).

### Long-Term Followup

The children were between 21 months and 11 years at the time of their developmental followup. The youngest child was given the Bayley Scales of Infant Development<sup>7</sup> and the older children were given the Stanford-Binet Intelligence Scale.<sup>8</sup> All of the children were functioning well within normal limits; the average IQ was 101 (Table 3).

With the exception of the youngest child, all of the children were also growing normally (Table 3). It should be noted that the child with the height and weight below the third percentile has always been small albeit healthy. Both of her parents are very short, and her short stature and low weight are not felt to be related to her heart disease at the present time.

Although the clinical outcome has been very good for these children; the psychological impact of this condition has been somewhat disappointing. Three of the set of parents are divorced, in part due to the overwhelming responsibility to raise a chronically handicapped child with risk of sudden death due to a malfunctioning pacemaker.<sup>10</sup> In repeated interviews with the parents we have learned about manipulative behavior and secondary gains by their children. In contrast, however, Galdston<sup>9</sup> has reported that children with pacemakers and their families are singularly free of emotional disturbances.

### Summary

In the early detection and aggressive management of children with congenital heart block and the children's increasing survival, low morbidity and

Table 2. Pacemaker Implants in Four Children with Congenital Heart Block.

Case	Pacemaker	
	Transvenous	Permanent
1	None	Placed at 10 mos. Revised at 3 and 5 yrs of age
2	Placed on 1st day and discontinued on day 6.	Placed at 3 yrs. Revised at 4 yrs.
3	Placed on day 2 and discontinued on day 10.	Place at 10 mos. Revised at 5 yrs.
4	Placed on day 8 and discontinued on day 27	Placed on day 29.

Table 3. Growth and Development Status of Four Children with Congenital Heart Block.

Case	Age (years)	Intelligence Quotient	Height (%)	Weight (%)
1	11	100	50	75
2	4 11/12	94	50	50
3	4 5/12	102	10	10
4	1 9/12	108	<3	<3

normal growth and development are now realities.<sup>11-13</sup> However, we must continue to be sensitive to the psychological impact that living with a pacemaker can have on the child and his family. A close cooperation between the cardiologist, cardiac surgeon, primary physician and parents is essential, to foster an adequate environment for these children and thus promote a productive adulthood.

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# LETTERS

## Fairfax data show no consistent trend in ampicillin-resistant *H influenzae*

Primary care physicians in Virginia must be concerned about trends in the prevalence of resistance to ampicillin in *Hemophilus influenzae* meningitis, and no doubt many read with interest the article on the subject by Armstrong and Butler in a recent issue of VIRGINIA MEDICAL.<sup>1</sup>

We previously reported data on patients with *H influenzae* meningitis managed at Fairfax Hospital from 1976 to 1982<sup>2,3</sup> and now present data for such patients admitted during 1983 as well. Our data

differ somewhat from those reported by Armstrong and Butler, and so we believe it is worthwhile to bring our findings to the attention of Virginia physicians.

The figures in the table show the number of patients admitted to Fairfax Hospital for management of *H influenzae* meningitis in the years 1977-1983; the percentage of those patients from whom ampicillin-resistant *H influenzae* were recovered from CSF specimens.

**Incidence of Ampicillin-Resistant Strains of *H. influenzae* Meningitis in Patients at Fairfax Hospital 1977-1983.**

	1977	1978	1979	1980	1981	1982	1983
Total patients with <i>H influenzae</i> meningitis	12	12	13	21	20	21	9
Ampicillin-resistant organisms from CSF specimen	0%	33%	15%	24%	20%	33%	11%

In Fairfax County, there has been no consistent trend from 1977 to 1983 in the percent of CSF isolates from meningitis patients containing *H influenzae* resistant to ampicillin. For instance, only one of the nine CSF isolates obtained in 1983 was resistant to ampicillin, while in 1982 seven (33%) of 21 CSF isolates of *H influenzae* organisms from patients with meningitis were ampicillin-resistant.

There are regional, as well as periodic, differences within Virginia in the prevalence of meningitis caused by *H influenzae* resistant to ampicillin.

**Richard H. Schwartz, MD**  
**Robin I. Goldenberg, MD**  
 Choong Park, PhD

The Fairfax Hospital  
 3300 Gallows Road  
 Falls Church VA 22046

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**The authors reply:** Local differences in the prevalence of resistance undoubtedly exist in Virginia as they do nationally. All we can say is that for the regions we examined, there were no statistically significant differences.

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# ABSTRACTS

*These are abstracts of papers to be presented at the annual meeting of the Virginia Surgical Society at the Homestead on April 28-29. Dr. H. H. Newsome, Jr., is program chairman.*

**Down the GI Tract from A to Z in Reverse (Zenker's to Anus).** W. Levi Old, Jr., MD, *Norfolk*.

Eponyms are here to stay. This presentation is a brief history of surgery of the esophago-gastro-intestinal tract featuring slide pictures of the title page of the major original yesteryear publications of Zenker, Heller, Mallory-Weiss, Billroth, Mikulicz, Whipple, Crohn, McBurney, Miles, et al. A brief biographical sketch and a picture of each of the 25 pioneers is shown.

**Obstruction and Perforation: High Risk Factors to Consider in the Staging of Colorectal Carcinoma.** J. W. Milsom, MD, and H. J. Wanebo, MD, *Charlottesville*.

Obstructing and perforated cancers of the colon are known to be associated with increased morbidity and mortality when compared to non-obstructed, non-perforated lesions. However, the American Joint Committee on Cancer staging does not recognize these entities in the staging of colorectal cancer. We have therefore examined this issue in a retrospective review of patients with colorectal adenocarcinoma treated at our institution over the past decade (1970-1980). Fourteen of 537 patients with the diagnosis of colorectal cancer presented with either perforation or obstruction. There were no Duke's A lesions which obstructed or perforated. Seven of the fourteen were Duke's B lesions, the other half being Duke's C lesions. Of the perforated/obstructed Duke's B patients, 1 of 7 (14%) were alive at 5 years. None of the perforated/obstructed Duke's C patients survived 5 years. In contrast, the remaining Duke's B patients in the study showed a 50% survival, and remaining Duke's C patients demonstrated a 19% 5 year survival. Local recurrence and intra abdominal implants were common occurrences in the patients with obstructing or perforated tumors. These figures are comparable to similar studies in the literature. Obstruction and perforation are poor prognostic signs for the patient with colorectal cancer and have a negative impact on survival beyond that expected with tumor stage per se. These should be included in American Joint Committee on Cancer

Stage Classification (TNM system) for colorectal cancer.

**The Clinical Utility of Indium<sup>111</sup>-Labeled White Blood Cell Imaging in the Surgical Patient.** J. Richard Hobson, MD, Robert E. Berry, MD, Marshall A. Wakat, MD, and Robert W. Beightol, PharmD, *Roanoke*.

White blood cells labeled with indium<sup>111</sup> have been used for the detection of localized inflammatory disease, especially abscesses. We have performed these studies in a community hospital setting since August, 1983. Over 50 patients have been studied. A simple labeling technique using commercially available indium<sup>111</sup> oxine, which yields high labeling efficiency and reliable white blood cell viability, will be described. In addition to intense white blood cell uptake in inflammatory and infectious disease, we have seen several patients with intense leukocytosis in necrotic areas of tumor masses. To date our sensitivity has been 92% and specificity has been 92%. False negative scans have occurred when abscess sites were obscured by normal activity from the liver and spleen. The judicious use of computer-assisted liver-spleen subtraction from the indium<sup>111</sup> white blood cell images improves the accuracy of studies for disease in or about the liver and spleen. White blood cell imaging is felt to be superior to gallium<sup>67</sup> image quality because of lack of interference from contaminated feces and lack of uptake in normal postoperative wounds. Clinically, the study has been most useful in abscesses suspected in the postoperative period, possible graft infections, necrotizing pancreatitis and ischemic, infected, or inflamed bowel.

**Combined Drainage for Pancreatic, Biliary and Gastric Obstruction in Chronic Pancreatitis.** G. R. Barnhart, MD, D. Margolius, MD, and H. J. Sugerman, MD, *Richmond*.

The results of simultaneous, combined pancreatic, biliary and gastric drainage procedures for obstructions secondary to chronic pancreatitis were reviewed. Between 1979 and 1983, 7 patients underwent multiple simultaneous drainage procedures for



chronic alcoholic pancreatitis. There were 5 males and 2 females ranging in age from 36 to 44 ( $40 \pm 4$ ) years old. No patient had ascites or laboratory evidence for hepatic dysfunction. The average common duct measurement by ultrasound in 6 patients was  $12.4 \pm 4.9$  mm. ERCP in 5 patients demonstrated pancreatic duct enlargement in each patient and biliary stricture in 4 patients. One patient had portal venous obstruction secondary to chronic pancreatitis with extensive peri-pancreatic venous collaterals. One patient had common duct obstruction secondary to pancreaticolithiasis. All patients underwent a longitudinal pancreaticojejunostomy, 5 patients underwent a choledochoduodenostomy and 2 patients a choledochojejunostomy. One patient had an associated duodenal stricture and also underwent a gastrojejunostomy. The total bilirubin fell from  $3.3 \pm 6.9$  to  $0.4 \pm 0.2$  mg/dl and alkaline phosphatase from  $481 \pm 402$  to  $118 \pm 35$  U/l ( $p = 0.055$ ) at 2 to 48 months (mean 21 months) following surgery. The abdominal pain was completely relieved in 3 patients and markedly improved in 2 patients. The 2 patients who had flow of bile from the choledochojejunostomy over the pancreatojejunostomy were asymptomatic. One patient who had persistent pain postoperatively was found to have ampullary carcinoma at endoscopy and underwent pancreaticoduodenectomy 5 months later. We conclude that multiple simultaneous drainage procedures for pancreatobiliary obstruction secondary to chronic pancreatitis is associated with a low morbidity, improved clinical condition and preferred to resection in the management of these patients, but that continued late evaluation is mandatory.

**Continuous Hepatic Artery Infusion Chemotherapy for Metastatic Colorectal Cancer.** George C. Hoffman, MD, James J. Stark, MD, Claiborne W. Fitchett, MD, R. Cecil Chapman, MD, John W. Baker, Jr., MD, and Alvin Margolius, Jr., MD, *Norfolk*.

Continuous infusion of chemotherapeutic agents into the hepatic arterial circulation for the treatment of metastatic colorectal cancer has been used in the past with varying success. A totally implantable infusion pump is now available for this purpose and has been used by others with encouraging results.

Eight patients with metastatic colorectal cancer to the liver underwent placement of an implantable infusion device for continuous hepatic arterial chemotherapy. One patient with severe hepatic involvement by tumor had progressive hepatic deterioration in the postoperative period and died of liver

failure two months after surgery without receiving significant intra-arterial chemotherapy.

In the seven remaining patients, six developed one or more complications of chemotherapy. These included systemic side effects of chemotherapy, hepatic toxicity, and gastritis. Most side effects were responsive to temporary discontinuing or changing the patient's chemotherapy program. One patient developed extensive antral gastritis which did not improve after stopping the chemotherapy and with aggressive medical therapy, and this patient required a gastric resection. Another patient with a permanent colostomy developed a pump pouch infection which was managed with oral antibiotic therapy and repeated pouch aspirations.

Six of the seven patients had significant tumor regression in the liver as defined by two or more criteria including liver scan, CAT scan, CEA, or improvement in their physical exam. Six of the seven patients developed extra-hepatic recurrence within 3–13 months after initiation of infusion therapy and four of the seven succumbed to extra-hepatic disease within 5–13 months.

Our experience with continuous hepatic artery and infusion chemotherapy using an implantable pump would suggest that this is an effective mode of therapy for controlling progressive hepatic metastases from colorectal cancer, but it is not without significant complications, and the progression of extra-hepatic disease is distressingly frequent and of rapid occurrence in the majority of patients.

**Emergency Room Thoracotomy: A 26-Month Experience at a Tertiary Trauma Center.** C. William Schwab, MD, and O. T. Adcock, MD, *Norfolk*.

Emergency room thoracotomy (ERT) was used during a 26-month period as a salvage procedure in severely injured patients who met the following criteria: a) a penetrating chest injury with no recordable V/S; b) penetrating or blunt trauma with witnessed signs of life (heartbeat, pulse, respirations or speech) then subsequent deterioration and arrest; and/or c) irreversible hypovolemic shock. To determine the frequency and outcome of ERT, the records were reviewed from January 1981 to March 1983.

All ERTs were performed under a standard protocol of left anterior thoracotomy through the fifth intercostal space, release of cardiac tamponade, or open cardiac massage with aortic cross clamping as indicated. If the patient became stable, definitive operative procedures were continued in the operating room.

Fifty-one ERTs were performed, 50 adults and 1

child. Thirty-six (71%) were for penetrating injuries; 18 stab wounds and 18 gunshot wounds. Fifteen (29%) ERTs were performed for blunt injury. The overall survivability was 27.5% (14 patients) and all were from the penetrating group (13 stab wounds, 1 gunshot wound). Signs of life were temporarily restored in an additional three patients, all of whom ultimately died. Stab wounds of the chest with cardiac laceration and tamponade accounted for one-third (18 patients) of our series and had the largest number of survivors: 13 (72.2%). Only one bluntly injured patient had signs of life restored long enough for transfer to the operating room.

We conclude that ERT is an acceptable procedure to salvage agonal patients with penetrating injury; it must be performed under a protocol that allows some selectivity so useless procedures are avoided. ERT in blunt agonal trauma at this time seems futile but requires further study.

**Optimal Management of Lower Extremity Skin Grafts.** Larry S. Nichter, MD, Raymond F. Morgan, MD, Jed H. Horowitz, MD, and Milton T. Edgerton, MD, *Charlottesville*.

Postoperative management of patients with lower extremity skin grafts traditionally requires prolonged bed rest and leg elevation. Complications from this treatment plan include deep venous thrombosis, stiff joints and prolonged inactivity. The use of silastic foam elastomer conformer in conjunction with overlying elastic compression provides optimal management. This method permits earlier ambulation, decreased hospitalization and few complications.

**Early Success with Tibial Vein Grafts for Limb Salvage.** Jesse Davidson, MD, Robert Keeley, MD, Philip Kistler, MD, Thomas Henretta, MD, and David Minichan, MD, *Roanoke*.

Autogenous saphenous vein bypass to the tibial arteries has become an effective method of limb salvage in the severely ischemic lower extremity. This report evaluates 30 patients who have undergone 35 infra-popliteal artery bypass grafting procedures with saphenous vein in the period July 1982 through February 1984. Fifty percent of patients had pre-existing diabetes mellitus. All patients underwent operation for limb salvage. Rest pain was the indication in 14 procedures, and 21 were performed for tissue necrosis. The posterior tibial artery was the recipient vessel in 20 cases, the anterior tibial in 11 cases, the peroneal in 4. Intraoperative arteriography was performed in selected

instances. Thirty-day patency was 91%. Early thrombectomy was necessary in two patients; however, the grafts remained patent. There was one perioperative death. Two major amputations have been performed, both secondary to severe diabetic foot infection despite graft patency. The importance of patient selection and operative technique is emphasized.

**Prognosis of Level V Malignant Melanoma.** Harry D. Bear, MD, James P. Neifeld, MD, and Saul Kay, MD, *Richmond*.

Level V melanomas account for 4%–8% of patients presenting with Stage I and II disease in many series with 5-year survival rates reported to be less than 20%. In-depth analysis of Level V melanomas has not been previously reported.

From 1952 through 1975, 40 patients presented with primary Clark's Level V melanomas. Twenty-one patients presented with clinical Stage I disease, 18 with Stage II, and one patient with distant metastases. Among Stage I patients, 10 were treated by wide excision alone and 11 underwent wide excision plus prophylactic regional lymph node dissection (RLND); 7 of 11 patients had histologically positive nodes. Ten Stage II patients were treated by wide excision and RLND, 3 underwent hemipelvectomy, and 5 refused surgery. Among 25 evaluable patients with adequate follow-up, actuarial 5-year survival was 45%. For Stage I patients, survival was 58% and disease-free survival (DFS) 36% at 5 years. Six of 10 occurrences were local or regional only. Prophylactic RLND did not appear to influence recurrence or survival rates. Following wide excision alone, 3 patients subsequently underwent RLND for regionally metastatic melanoma and 2 have remained free of disease. Among 11 evaluable Stage II patients, overall survival was 58% and DFS 45% at 3 years. Of 5 patients who had subsequent recurrences, 4 had distant metastases. All 9 patients with distant metastases at the time of first recurrence died of disease within 14 months (median 3 months) of detection of metastatic disease. Primary melanomas of the foot (10 patients) and trunk (4 patients) had a worse prognosis than other sites. Ulceration (seen in 10 of 25 patients) did not appear to significantly influence outcome.

These data suggest that most patients with Level V melanoma present with clinically localized disease. Prophylactic RLND did not significantly affect disease-free or overall survival. The invasiveness of these deep tumors appears to reduce the influence of other factors, including nodal status. The prognosis of patients with Level V melanoma,



even with clinically or histologically positive lymph nodes, is not hopeless and these patients should be treated aggressively.

**Bile Duct Reconstruction with Transanastomotic U Tubes.** George A. Parker, MD, and L. Gregg Halloran, MD, *Richmond*.

Thirty patients underwent bile duct reconstruction or bypass utilizing transanastomotic U tubes to stent the bilo-enteric anastomoses. The U tubes are brought through the substance of the liver into the proximal bile duct, through the anastomosis, usually to Roux-en-Y limb of jejunum, and then withdrawn from the jejunum through a separate enterotomy. Both ends of the silastic catheters are then brought out the abdominal wall through separate stab incisions and secured to the abdominal wall. Depending upon the number of bile ducts to which anastomoses must be performed, as many as three U tubes have been utilized.

In 20 patients the procedure was performed for benign disease. There were no operative deaths. There were 5 infectious complications, none of which were life-threatening. Preoperative bilirubin levels averaged 10.4 mg %; postoperative bilirubin levels averaged 2.8 mg %. Recurrent strictures developed in 2 patients at 20 and 19 months following removal of the U tubes.

In 10 patients the biliary obstruction was secondary to malignant disease. Seven patients had primary biliary tract tumors; three had metastatic tumors. Operative mortality was 20%. Among the operative survivors the preoperative median bilirubin level was 10.2 mg % (range 5.1 to 27.6 mg %) and the postoperative level was 1.5 mg % (range 0.2 to 10.7 mg %). Three patients developed abdominal abscesses; two of them died. Another patient developed an abscess in one U tube tract which responded to drainage.

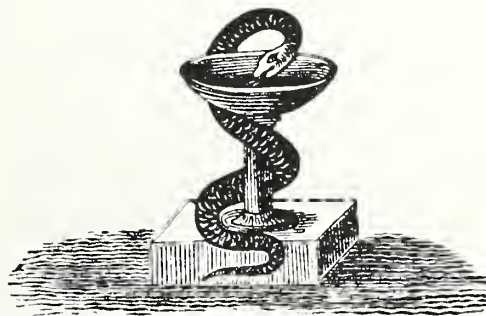
The use of transanastomotic U tubes successfully

stents difficult bilo-enteric anastomoses. The ease with which the tubes can be changed if they become clogged and the patient acceptance of them makes the placement of transanastomotic U tubes the procedure of choice when reconstructing or bypassing proximal bile duct obstruction.

**Recurrent Pressure Sores: Evaluation and Treatment.** John G. Kenney, MD, Milton T. Edgerton, MD, and Richard F. Edlich, MD, *Charlottesville*.

Coverage of recurrent pressure ulcer remains a challenging clinical problem since the first pedicle flap is usually unsuitable for advancement and a new flap must be constructed. A variety of salvage procedures using myocutaneous flaps of different design was employed successfully in 20 patients with recurrent pressure ulcers. The primary ulcers of these patients were first treated by debridement and primary ostectomy and then coverage by either skin grafts, skin flaps or myocutaneous flaps.

Surgery was undertaken in these patients when they were (1) immunocompetent as evidenced by delayed hypersensitivity skin tests; (2) had a positive nitrogen balance; and (3) had a wound bacterial count below  $10^5$  bacteria/gm tissue and had no evidence of severe spasticity that interfered with rehabilitation. Ten patients with recurrent ischial ulcers initially covered by either a gluteus maximus or gracilis myocutaneous flap were reconstructed with either hamstring V-Y advancement flaps or vastus lateralis myocutaneous flaps. A biceps femoris or vastus lateralis myocutaneous flap was employed in 6 patients with recurrent trochanteric pressure sores that were initially covered by tensor fascia lata myocutaneous flaps. Four patients with recurrent sacral ulcers first covered with either a skin graft or gluteal skin flap were reconstructed by either a gluteus maximus or tensor fascia lata myocutaneous flap.



# VIRGINIA Medical Monthly.

LANDON B. EDWARDS, M. D.,

*Member of the Va. State Board of Health, Recording Secretary Medical Society of Virginia, &c.,*

Editor and Proprietor.

EDO UT PROSIM.



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RICHMOND, VA.



## A Case of Fractured Feelings

**T**HERE are some lessons in medical practice that can only be learned the hard way, and for some problems there are no lessons at all, neither in medical school nor through experience.

How, for instance, do you deal with a colleague or former teacher to whom you have referred a patient but with whose diagnosis or management you disagree? One such incident in my own practice still troubles me, although I know of nothing I would do differently today.

I had not long been in practice when a patient, a very sweet elderly lady, came in complaining of chest pain which followed descending her basement steps, not climbing them. This pain was centered in the left lateral chest, had persisted three hours, was not related to exertion, not associated with diaphoresis, cough, hemoptysis or dyspnea.

The little lady was about 70 years of age, had never smoked in her life nor had asthma or any other pulmonary symptoms other than the one episode of chest pain on descending the basement stairs. EKGs were normal, and the remainder of the physical exam was unrevealing except for extensive osteoarthritis in multiple joints, most painful and stiff in the hips and knees. She was bright, cheerful, with a keen sense of humor, and overall just a very charming and down-to-earth person, the adored

mother of six or eight children, all grown. Family history was essentially negative except for one son who had died of lymphoma several years previously.

Chest fluoroscopy, which we did in those days, showed a hazy infiltrate in the left mid-lung field. I did not have a chest x-ray machine in the office at that time, and she had little money, so I sent her to the county health department, where chest x-rays were done inexpensively, or free, and I later went to the health department to review the film.

As expected, there was a soft infiltrate in the left mid-lung field, roughly  $2 \times 10 \times 6$  cm, triangular in shape with apex toward the hilum, and not quite extending to the periphery of the lung. It was certainly not solid and on first impression most likely suggested pulmonary embolus, although there was none of the usually associated signs or symptoms.

This film was carried to a well-known thoracic surgeon who was also a professor. He agreed that it suggested a pulmonary embolus but was also at a loss to explain the absence of expected signs and symptoms from a lesion this large. I asked if he thought that it could be a malignancy, and he said Yes, it might be, but the probability was very low because the patient had none of the usual findings.

He suggested that we repeat the film in a month to see if the shadow got larger or smaller.

We did repeat it in a month, and indeed the shadow had gotten larger and denser. Her chest pain had cleared spontaneously, she was symptom-free, EKGs were normal. The surgeon-professor now felt it was probably a malignancy, and after a negative bronchoscopy suggested a thoractomy and probably either a pneumonectomy or segmental resection.

The patient agreed to surgery and successfully underwent resection of the malignancy in the left mid-lung field. I never received a pathology report or any other report from the surgeon but was told that it was a "small cell tumor." The patient did well post-op and was discharged home, and when I saw her in the office, she complained only of the expected post-op pain, a "stitch", as she called it, jokingly accusing the surgeon of leaving his thimble in her chest. This gradually regressed, and she did well until five or six weeks later, when she again developed chest pain, but now on the right side, opposite the surgical site. This time chest x-ray revealed an infiltration in the right lower lung field. I carried the film promptly to the thoracic surgeon to ask his advice.

He said the patient now obviously had metastatic disease into the right thorax and that she was not a candidate for any further surgery. I gently suggested that it looked like pneumonia to me and that I proposed treating her with penicillin, which I did,

and the "metastasis" promptly cleared, as did a low-grade fever. This brought no comment from the surgeon. I asked him to send me the pathology reports in order to write up the case, for I knew there was a valuable lesson in it for me and perhaps for others.

Months passed, and no pathology or operative report or any other communication was ever forthcoming. I asked again for them, but they were never received. Years later I asked the record room several times to send them to me but never received them—and still haven't, after 30 years.

Over the next ten to 15 years the little old lady did beautifully, except for her relentless arthritis and developing PA, which responded nicely to B-12 shots. About 18 years later she developed complete heart block on a visit to Florida and did well with a pacemaker, but she was lost to followup when she moved to Texas. I received Christmas cards with progress notes for a couple of years, but then heard nothing, and I must assume the infirmities of age finally did her in.

My mentor in the case is now dead. It appears that I irreparably fractured his feelings, and for that I am sorry. But I still do not know how I could, or would, have done differently. Responsibility to the patient is paramount, feelings be damned!

ROBERT L. HOWARD, MD

2745 North Radford Street  
Arlington VA 22207

## The Resident Physician Section

**M**EMBERSHIP in The Medical Society of Virginia is integral to participation in organized medicine. To me, that makes MSV membership an opportunity not to be missed. I do not believe the skeptics who view organized medicine as a doctors' labor union or who disdain the "politics" of organized medicine.

Organized medicine does not work solely for the benefit of doctors. For example, organized medicine fights constantly to preserve the excellent quality of American medicine, by opposing such threats to quality of care as excessive governmental regulation. The benefactors of this effort are patients and taxpayers, i.e., the American people.

As for politics, organized medicine is a democratic system, with participation by all members the key to accurate representation of the consensus. I cer-

tainly want to be there when the decisions are made so that I can affect that consensus.

The Society's new Resident Physician Section offers entry to organized medicine's democratic process. It offers a chance to learn to lead, by learning to follow, and to begin to understand, by hearing the issues, the many complex problems confronting medicine today.

I think it is imperative that residents and other young physicians get in on the ground floor of organized medicine now, so that we can contribute positively to the shape of medicine to come.

SAM BARTON, MD

820 South Main Street  
Blackstone VA 23824

Dr. Barton is a resident in family practice at the Blackstone Family Practice Center.



# VIRGINIA MEDICAL OBITUARY

## H. J. Warthen, MD

Dr. Harry Justice Warthen, Jr., surgeon, historian and editor, died March 5 at his Richmond home. He was 82 years old and had long suffered from emphysema.

At a meeting less than a year ago, the Richmond Academy of Medicine paid tribute to Dr. Warthen for the amazing diversity of his accomplishments. Dr. Warthen had been president of the Academy. He was Editor of this journal for 20 years. A prolific editorialist, he also wrote many works of history that are an enduring contribution to medicine and to Virginia, his native state. The Medical Society of Virginia honored him with its Physician Award for Community Service. Richmond Memorial Hospital claimed him as a founder and its first chief of surgery. For his alma mater, the University of Virginia, he had been president of the Medical Alumni Association.

Dr. Warthen's death occurred as this issue was being made up for the printer, precluding a full account of his distinguished lifetime. That will appear in a future issue.

## Solomon N. Albert, MD

Dr. Solomon Naphtali Albert, Arlington, died October 11, 1983, at the Washington, DC, Hospital Center, where he had been director of the anesthesiology research laboratory. He was 67.

Born in Beirut, Lebanon, Dr. Albert was graduated in 1940 from the American University of Beirut medical school. For his service with the French Army during World War II he was named a chevalier of the French Legion of Honor. He took his training in anesthesiology in England, then came to the States to fulfill a fellowship at George Washington University Hospital in Washington and made his home in Arlington thereafter.

The author of three texts and many technical articles, Dr. Albert was co-developer of the Albert-Swan dilution computer used in determining blood volume.

Dr. Albert came to membership in The Medical Society of Virginia in 1956 through the Arlington County Medical Society. His other professional memberships included the Royal College of Sur-

geons, Royal Society of Medicine, American College of Anesthesiologists, American College of Nuclear Physicians, and the International Anesthesia Research Society.

## C. Fallon Davis, Jr., MD

Dr. Charles Fallon Davis, Jr., longtime Roanoke family practitioner, died February 3 at the age of 74.

A native of Radford, Virginia, Dr. Davis was graduated in 1934 from the Medical College of Virginia. He received training at St. Mary's Hospital, Huntington, West Virginia, and at Mercy Hospital in Baltimore, Maryland. From 1940 to 1946, Dr. Davis served in the US Army as a flight surgeon, and following active duty, continued to serve in the Army Reserve until 1973, when he retired a colonel. In 1947, he established the Melrose Clinic in Roanoke, practicing there for the rest of his life.

Dr. Davis had been a member of The Medical Society of Virginia and the Roanoke Academy of Medicine for over 36 years. He belonged also to the American Academy of Family Physicians and the American Medical Association.

A son, Dr. Richard W. Davis, practices medicine in Johnstown, Pennsylvania.

## V. K. Thoppay, MD

Dr. Vasudevan K. Thoppay, a psychiatrist at Central State Hospital in Petersburg, died January 22 at the age of 44. Born in India, Dr. Thoppay was graduated from the Stanley Medical College of Madras University in Madras and came to this country in the early 1970s. His memberships included The Medical Society of Virginia and the Southside Medical Society.

## T. M. Arrington, MD

Dr. Thomas Marshall Arrington, general practitioner in Richmond for almost 30 years, died February 9. He was 64 years old.

A native of Ninety Six, South Carolina, Dr. Arrington was a graduate of the University of Florida and the Bowman Gray School of Medicine,

Wake Forest University, Winston-Salem, North Carolina. From the time he graduated from medical school until the end of World War II, he served in the Army Medical Corps. He entered private practice in Richmond in the 1950s, and from 1962 until 1981 was a case determiner at the Social Security Disability Office, but returned to private practice before retiring in 1981.

Dr. Arrington came to membership in The Medical Society of Virginia through the Richmond Academy of Medicine.

## Memoir of W. H. Nicholson 1913-1984

*By Charles S. Miller, MD*

Dr. William Henry Nicholson, aged 70, of Elkton, Virginia, died January 28, 1984. He was born September 10, 1913, in Wilkes Barre, Pennsylvania, the son of the late Frank Craig and Henrietta Hines Nicholson.

Dr. Nicholson was graduated from the Pennsylvania State University at University Park in 1935 and from Temple University School of Medicine in Philadelphia in 1939. He interned at Germantown Hospital in Philadelphia.

In 1941 he went to work for Merck and Company and was plant physician at Merck's Stonewall plant in Elkton for 36 years until he retired in 1978. He then conducted a private practice until failing health forced him to discontinue in 1980.

He was a member of The Medical Society of Virginia, American Medical Association, Rockingham County Medical Society and American Academy of Family Physicians and was an honorary member of the staff of Rockingham Memorial Hospital.

Dr. Nicholson is survived by his wife, Lillian Rupert Nicholson; two sons, Frank C. Nicholson and William H. Nicholson; a daughter, Mary Louise Nicholson; a sister, Rhea Nicholson Tench; and four grandchildren.

"Doc," as many people called him, had many hobbies. Perhaps first among these was his love of horses. He was also a breeder of beef cattle. He was an avid lover of sailboating and sailed his boat at every opportunity as long as his health permitted. He had served as a deacon and trustee of the Elkton Presbyterian Church.

He served his profession and community long and well and will be sadly missed.

## Memoir of J. B. Funkhouser 1913-1983

*By James O. Burke, MD,  
H. St. George Tucker, MD,  
and R. Campbell Manson, MD*

James Bauer Funkhouser, born in Indianapolis 11 July 1913, son of a physician, died 24 January 1983 in Richmond. He was educated in Indianapolis schools and at the University of Indiana, from which he received the BS degree in 1935 and MD degree in 1937. His psychiatric training was at Southwestern State Hospital in Marion, Virginia, and New York Psychiatric Institute.

During World War II he served with distinction in the US Army in Africa, Italy and Vienna. Much of that time he was with the 45th General Hospital, the McGuire-MCV Unit, where he was the chief of the neuropsychiatric service. He received the Army's Bronze Star for achievement. Following retirement as a major, he located in Richmond, becoming chief of neuropsychiatry at McGuire Veterans Hospital and an associate clinical professor of psychiatry at the Medical College of Virginia.

From 1957 to 1979, Dr. Funkhouser served the Commonwealth of Virginia as assistant and later Deputy Commissioner of the Department of Mental Health and Mental Retardation. Originally, when asked to join that department as the Commissioner, he declined, stating he would become Assistant Commissioner if Dr. Hiram Davis, a former psychiatric resident of Dr. Funkhouser's, were the Commissioner. His request was granted and during his time in that department Dr. Funkhouser served as mental hygiene program consultant and Director of Training and Research. The development of the mental hygiene clinic system is the assignment of which he was the most proud. There were expansions of all clinics and development of new ones, increasing the total from 12 to 30. He also served as Acting Superintendent at various times of Eastern State Hospital, Central State Hospital, Western State Hospital, Southwestern State Hospital and the Southern Virginia Psychiatric Institute in Danville.

He was a member of the Governor's Overall Advisory Committee on Needs of Handicapped Children, the Virginia Council for the Deaf and the Governor's Hospital Advisory Council. He was a member of the American Academy of Neurology, American Psychiatric Association, Neuropsychiatric Society of Virginia, serving as its president, Association for Research of Nervous and Mental



Diseases, The Medical Society of Virginia and the AMA. He was certified by the American Board of Psychiatry. Developing an interest in epilepsy, he studied electroencephalography in Chicago, was certified in that subspecialty and became widely known as one of the most experienced electroencephalographers in the Commonwealth.

As a man, a person, a friend and a wartime companion, Jim Funkhouser was humorous, warm and attractive; a student of literature, he often quoted Shakespeare, Dickens, T. S. Elliot, Kipling. He was a special devotee of James Branch Cabell. He had an unusually fine voice and frequently brought us out of the war's depressions by his songs and encouraged us to sing along with him. He learned Italian well enough to speak it as well as sing the Italian operas. An accomplished musician, he played the guitar and banjo. He was a chess player, golfer and sculptor. He had been a member of the Grace and Holy Trinity Episcopal Church choir.

He is survived by his wife, Mrs. Gene Smith Funkhouser, two daughters, Mrs. George H. Flowers and Dr. Laura S. Funkhouser, all of Richmond, and a son, Dr. John J. Funkhouser of West Falmouth, Massachusetts, six grandchildren and a sister, Mrs. Alan Johnstone of Martinsville, Indiana.

Prepared and published at the request of the Richmond Academy of Medicine.

## Memoir of A. C. Wyman 1918-1983

*By Ira J. Green, MD*

Dr. Alvin Charles Wyman died unexpectedly from heart disease on March 28, 1983, several hours after beginning work that day at the George Washington University Hospital. Dr. Wyman's professional career strongly influenced two spheres of radiological practice: the teaching program in the Department of Radiology at George Washington University Medical Center in the latter portion of his life and, earlier, the development of a sophisticated radiology practice in Alexandria, Virginia.

Dr. Wyman was born in New York City on April 13, 1918. He obtained his BA from Cornell University in 1939 and his MD from the New York University College of Medicine in 1943. He did his internship at the United States Naval Hospital in Norfolk, Virginia, and his residency training in

radiology at the United States Naval Hospitals in Bethesda, Maryland, and Long Beach, California. He served with the United States Navy from 1942 to 1957.

Throughout his radiological career, Doctor Wyman published extensively. A method devised by him using plain abdominal film for splenic size evaluation remained the definitive method for almost two decades. He co-authored anatomy texts which have become required references for radiologists.

The appointments and honors bestowed upon him include fellowship in the American College of Radiology and in the American College of Physicians. He was clinical professor of radiology at George Washington University School of Medicine from 1971 until his death. He was a past president of the Washington Metropolitan Chapter of the American College of Radiology and a councilor of that chapter until 1978.

He was a beloved teacher, of radiology residents, and everyone touched by his combination of enthusiasm, knowledge, and extraordinary ability to communicate became a better radiologist through him. It was Dr. Wyman who made enlightened radiology an integral part of medical practice in Northern Virginia. It is difficult to believe but true that it was only after 1947, when Dr. Wyman became chairman of the Radiology Department at Alexandria Hospital, that the scientific, consultative and analytic approach to radiological diagnosis became the standard in Alexandria. This was exclusively due to his efforts at the onset. His influence extended well beyond Alexandria hospital and those of us practicing in Northern Virginia today, not only in radiology but in all medical fields, are profoundly grateful to Dr. Wyman, for he was the generating force of the radiology we practice today.

While in Alexandria, he served as medical director of the McCluer Tumor Clinic from 1952 to 1970 and he made that cancer clinic for indigent patients a place where all received the best in evaluation and treatment available anywhere and where those of us who were privileged to participate learned lessons in humanism and medical science that made us better physicians. It was Dr. Wyman who created and nurtured this special place.

We remember Al Wyman with great affection, great respect and unending gratitude for what he did and the example that he set. His students and colleagues still feel him at their sides.

Prepared and published at the request of the Alexandria Medical Society.

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the Virginia doctors' classified.



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## Freezing fees is hot topic in Virginia press

News of the American Medical Association's recent appeal to physicians to freeze their fees for one year was deemed fit to print by newspapers all over Virginia, drawing mixed reviews from both the physicians whom reporters sought out for quotes and from editorialists. Out of the abundant lineage, here are some excerpts.

Under the title, "Freeze shows proper spirit," the *Charlottesville Daily Progress* concluded an editorial with these sentences: "Doctors earn more than enough to tighten their belts marginally without feeling pain. Those who agree to do so deserve credit, however, for doing it voluntarily. Professionals in other fields where fees are on the rise haven't been willing to make even a modest sacrifice."

The *Newport News Daily Press* quoted Dr. J. Travers Edwards, a private practitioner in Newport News: "Locally, it's very likely physicians will volunteer to [freeze fees] . . . Unfortunately, suppliers who supply x-rays and EKG tapes aren't going to freeze their prices, and doctors' overhead will rise," Edwards said.

"Doctors receptive to freeze," read the headline in the *Danville Register*, whose reporter talked to three Danville physicians.

"Certainly I'm willing to do anything I can to keep the cost of medical care down," said Dr. Syed Ahmed, who practices internal medicine and cardiology' . . .

"Dr. John J. Marsella, an obstetrician and gynecologist, said there is probably a certain number of

doctors who charge without considering a patient's ability to pay, 'but the vast majority of them make some kind of concession or another.' Marsella said the AMA appeal isn't unfair. 'Nobody thinks that's unusual because we've been doing it for so long,' he said.

"An eye, nose and throat specialist, Dr. F. H. McGovern, criticized the AMA for what he called a 'near-sighted approach to a great big problem.' He said if doctors freeze fees, they will also have to reduce service, and that will hurt the public.

"I'd freeze my wage if they'd freeze everything that went with it," McGovern said. 'Just my office drugs went up three times in cost in

one year. You can't operate a business with freezing your charges.' "

The *Norfolk Ledger-Star's* reporter called presidents of medical societies in the Tidewater area for their comments on a fee freeze and quoted them as follows.

"We're all for it," said Dr. Behrooz Dayanim, president of the Portsmouth Academy of Medicine.

"Dr. Lawrence Bernert, president of the Norfolk Academy of Medicine, called the AMA's suggestion 'a noble gesture,' but he said . . . 'I suspect doctors will continue their tradition of adjusting fees on a case-by-case basis as the need is demonstrated,' he said.

"Dr. John Krueger, a delegate

---

## First histoplasmosis death in a decade

For the first time in ten years, Virginia epidemiologists recorded late in March a death due to histoplasmosis. The victim was a young Petersburg man; nine others from that area fell ill of the fungal organism, several of them severely.

The young man who died was transferred from Petersburg General Hospital to the Medical College of Virginia Hospital, where Dr. Thomas M. Kerkerling of the Division of Infectious Diseases attended him. Later, on the MCV pediatrics service and at McGuire Veterans Administration Hospital, Dr. Kerkerling saw three other patients who seemed to have the disease. When he learned they were all from the same area, he alerted state health officials; they located the

other patients and the suspected source of infection, an old house being renovated in Dinwiddie County. All ten patients had been working on the house; they are thought to have acquired the disease from some debris contaminated by bird or bat droppings. Test results to confirm or deny the hypothesis were due late last month.

As a general rule, 10 to 12 cases of histoplasmosis are reported by Virginia physicians each year. Suspect histoplasmosis in a patient, Dr. Kerkerling advises, if a patient complains of fevers, night sweats and cough; if a chest x-ray shows interstitial infiltrate; and if the patient has an associated history of spelunking, or of disturbing an old bird roost, such as a chicken coop.



# NEWS BUREAU

from Virginia Beach to [The Medical Society of Virginia], predicted that doctors of the Virginia Beach Medical Society 'will by and large support the request.' Krueger added, however, that 'extenuating circumstances,' such as inflation, would make it difficult for doctors to 'independently and unilaterally hold the line' against increasing costs."

The president of the Chesapeake Medical Society, Dr. Juan Montero, was quoted by the *Norfolk Virginia Pilot* as saying that "'anything to keep the costs of health care down is welcome.' But [he] added that doctors' fees account for only 17% of health-care expenditures."

Four Lynchburg physicians discussed the proposed fee freeze with Cynthia T. Pegram of the *Lynchburg Daily Advance*.

"'Doctors are supposed to be and try to be advocates for patients in ways involving health and general welfare,' said R. Terrell Wingfield, a Lynchburg psychiatrist. . . . 'There's more to healing than drugs and surgery—concern for a person's total welfare is necessary.'

"Wingfield said he was 'very pleased and proud' that the AMA was 'visibly showing we have social and economic concerns for the people' . . .

"James A. Piggott, a pathologist at Virginia Baptist Hospital and current Lynchburg Academy of Medicine president, said. . . . 'I think physicians are leaders in the health care team, and probably many physicians will take the lead in this, trying to curb costs. I think a voluntary restriction is certainly more desirable than any government-imposed freeze.'

"Dr. Edward G. Calvert, a Lynchburg surgeon, said, 'I think it would be nice if, across the board, all professionals and businesses would (implement a) freeze . . .'

"Dr. David B. Hill, a thoracic surgeon, said that physicians must take the lead in cutting health care costs, and that the plan to freeze fees 'is a gesture of good intent . . . It doesn't amount to that much money. I think there are other connotations to this—the good will, the attention focused on health costs.' "

In Northern Virginia, "Some Area Doctors Can't Swallow AMA Prescription," said the headline in the *Washington Post*. The accompanying story quoted largely negative comments from physicians in the area, including: "'I don't know why doctors should be singled out,' said Dr. Louis Maciulla, an Arlington obstetrician. 'My nurses get a raise every year and expect that. And my overhead goes up. . . .'

## Rabies moving south

The outbreak of animal rabies in Northern Virginia reported in these pages in June 1983 is spreading southward, say state epidemiologists. In the newly affected rural areas the number of rabid animals reported has declined, however; 78 reports of rabid animals were tallied in the state from January through March of this year, as against 187 for the same period in 1983. This could change as the disease moves into the urban areas of Central Virginia. Raccoons continue to be the animals most often affected.

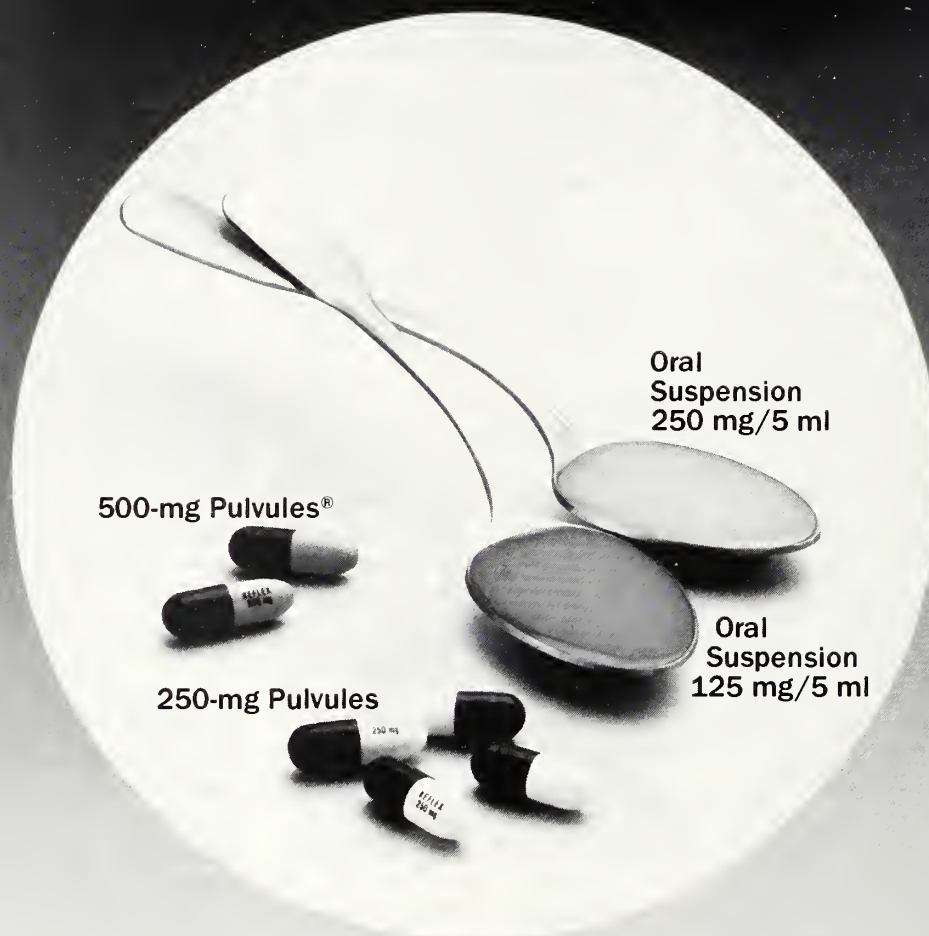
### 412 DELINQUENTS

Four hundred and twelve Medical Society of Virginia members have been billed four times for their 1984 dues. Are you one of these delinquents? If so, send in your check. You need your MSV membership, and the Society needs you.

The *Fredericksburg Free Lance-Star* referred to the AMA's freeze appeal in an editorial, "Paying the doctor," that dealt with Medicare costs and said in part, ". . . the Medicare system . . . encourages an escalator effect in doctors' fees that even the American Medical Association now suggests may need to be temporarily frozen. . . . There are other possible approaches. Doctors could be forbidden from charging Medicare patients more than the government-set rate. Medicare reimbursements could be temporarily frozen."

Freezing Medicare reimbursements was exactly what the United States House and Senate were wrangling about as this was written, with the House insisting on mandatory assignment as a corollary of the fee freeze and the Senate refusing to go along with mandatory assignment. Mailgrams went out from Medical Society of Virginia headquarters to Society officers and members of the Vanguard Committee, and Dr. Harry C. Kuykendall, MSV President Elect, took part in a "fly-in" of state society representatives who converged on the capitol to make House calls on their congressmen.

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## From the President:

**S**OMETIMES I can't help but wonder what happened to the image that the public had of doctors. What went wrong, and why?

Being from a small town in Virginia, where my father was one of the town's general practitioners, was something, or so I thought. He died when I was quite young, but I can still remember him getting up at night to go someplace on a call. If not that, I would see blood spots on the sidewalk where an injured patient had been brought to his office which was in our home. The nearest hospital was 20 miles away over a winding two-lane road.

After he died and I got older, everyone told me what a fine person he had been, a real pillar of the community. He was the kind who sent few bills and was paid if, and when, there was money. His estate was quite small and my mother went to work to educate my brother and me. My brother eventually went to law school and I went to medical school.

It wasn't until I had finished medical school that I discovered some people went to medical school for reasons other than the desire to practice medicine and to help others. I discovered the sad fact that some medical students had gone to school to avoid the draft or fighting part of the war. When it was over, they quit medicine and did other things. That was a shock and also an eye-opener for a small town southerner, I can tell you.

I started out to be a general practitioner, or perhaps a general internist, and I was advised by my godfather, Dr. William Mallory of Washington,



# Physician, Examine Yourself

to take a year of pathology, as it would make me a better physician. I took his advice and while training in Boston, Dr. Mallory suggested I stay in pathology, which I did. At times, I have been annoyed when a person had suggested that the reason I went into pathology was to make money. Money and medicine were two words that didn't go hand in glove as far as I was concerned, although I knew doctors lived comfortably.

In the last two decades, public concerns have grown because doctors seemed to make more money while appearing to spend less time practicing medicine.

As time went on, the doctors' parking lots became filled with bigger and more expensive cars. Vacations became more exotic and longer. House calls became a rarity.

Blue Cross and Blue Shield have certainly helped physicians get paid for things they hadn't been paid for before. Physicians also seemed to be in the forefront of efforts to oppose programs designed to help the poor and elderly, such as Medicare and Medicaid. It didn't matter that physicians may have found less expensive, but equally effective, alternatives. The perception was that we were opposed to the government helping to care for the elderly and the poor. But the advent of Medicaid and Medicare resulted in fewer and fewer no-pay patients. When such patients showed up, some physicians resented them taking up space, while others refused to accept Medicaid and Medicare payments.

Is it any wonder that our image has slipped as a whole, even though that of a patients' individual physician has not suffered to the same extent? Is it also any wonder that many politicians have felt less compelled to listen to organized medicine?

Now that DRGs, HMOs, PPOs, etc., are upon us, along with competition and a plethora of doctors, stronger incentives have been placed on all physicians to focus greater attention on how we treat patients. We're urged to minimize waiting time, exhibit more interest in the personal welfare of patients and their families, take time to listen, and be more open to suggestions about changing practice habits, such as more convenient office hours.

I also believe that the fee freeze advocated by the AMA deserves the support of all Virginia physicians. While I do not subscribe to the theory that physician fees are the primary cause of rising medical costs, I do believe it is important for physicians to demonstrate to the public their personal commitment to controlling health care costs.

There is no question that doctors will make less money and their income will be less guaranteed than previously. However, in the future, I'm sure few of us will starve and in the long run, our images may be improved. Those who went into medicine for altruistic reasons will still find it a wonderful and rewarding profession.

C. BARRIE COOK, MD, PRESIDENT,  
The Medical Society of Virginia

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**Practical Office Management of Chronic Disease** (Eastern Virginia Medical School), *Virginia Beach*. Office of CME, PO Box 1980, Norfolk VA 23501.

**July 14**

**Second Saturday Symposium: Cardiology** (Eastern Virginia Medical School), *Norfolk*. Office of CME, PO Box 1980, Norfolk VA 23501.

**July 22-28**

**Intensive Review of Internal Medicine** (University of Delaware), *Newark, Delaware*. Sylvia Brocka, 2800 Pennsylvania Ave., Wilmington DE 19806, (302) 451-8151.

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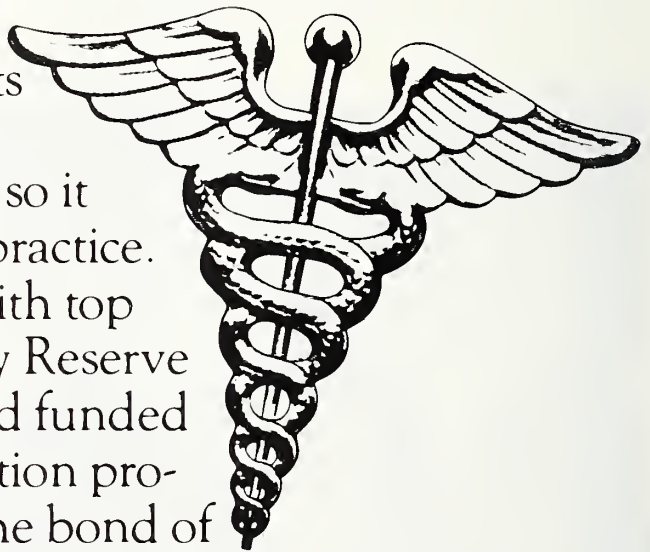
## Emissaries to a New Alliance

To strengthen solidarity in an era of divisive change, organized medicine is opening up new sections in its deliberative bodies. As the New Members list on page 259 attests, The Medical Society of Virginia's House of Delegates has a growing new Resident Physician Section, and at an American Medical Association meeting last year a new Medical Staff Section was launched. Pictured here are the

physicians who comprised the Virginia delegation to that new section. Seated, from left: Dr. Carlos E. Odiaga, Annandale; Dr. George J. Carroll, Suffolk; Dr. Carol S. Shapiro, Woodbridge; Dr. J. Shermer Garrison III, Portsmouth; and Dr. A. Reif Kessler II. Standing, from left: Dr. Raymond S. Jones, Fredericksburg; Dr. J. Hayden Hollingsworth, Roanoke; Dr. Harold L. Williams, Newport News; Dr. Herbert A. Wood, Winchester; Dr. Robert C. Garcia, Arlington; and Capt. Michael O. Murphy, MCUSN, Portsmouth.

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Box 159, UVa Medical Center, Charlottesville VA  
22908

## *Alleghany-Bath County Medical Society*

**Mary Jane Luke, MD**, Pediatric Cardiology, Box 351,  
Covington VA 24426

## *Fairfax County Medical Society*

**Russell C. Libby, MD**, Pediatrics, 316 Arlington Blvd.,  
Fairfax VA 22031

**Eugenia J. Quintos, MD**, Pathology, 6328 Mayapple  
Place, Alexandria VA 22312

## *Newport News Medical Society*

**Ray F. Rhodes, MD**, Anesthesiology, 30 River Road,  
Newport News VA 23601

## *Norfolk Academy of Medicine*

**Vincent R. Machaj, MD**, Anesthesiology, 948 Royal Oak  
Close, Virginia Beach VA 23452

## *Richmond Academy of Medicine*

**Mary Kay Dineen, MD**, Obstetrics/Gynecology, Box 34,  
MCV Station, Richmond VA 23298

**John E. Fitzgerald, MD**, Cardiology, 7601 Forest Ave.,  
Richmond VA 23229

**John R. Janes, Jr., MD**, Anesthesiology, 5855 Bremono  
Road, Richmond VA 23226

**Mickael M. Kannan, MD**, Infectious Diseases 5901 Lake-  
side Ave., Richmond VA 23228

**Grover C. Robinson III, MD**, Pediatrics, 23 Countryside  
Lane, Richmond VA 23229

**Mark S. Stensland, MD**, Family Practice, 2912 Fox Chase  
Dr., Richmond VA 23113

**Stewart J. Wetchler, MD**, Obstetrics/Gynecology, Box  
34, MCV Station, Richmond VA 23298

## *Roanoke Academy of Medicine*

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ley St., Roanoke VA 24018

**Roger P. Wiley, MD**, Radiology, 3557 Peakwood Dr.,  
SW, Roanoke VA 24014

## *Rockbridge County Medical Society*

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## *Southwestern Virginia Medical Society*

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## *Resident Physician Section*

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Blackstone VA 23824

**Thomas J. Carrico, MD**, Plastic Surgery, Box 154, MCV  
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**John G. Daniel, MD**, Family Practice, 201 College Ave.,  
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lottesville VA 22901

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Hare Dr., Richmond VA 23235

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Blvd., Norfolk VA 23517

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**Lynn C. Levitt, MD**, Family Practice, 2808 Sweetbriar  
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**Christine H. Llewellyn, MD**, Radiology, 3809 Gill St.,  
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**Carl D. Malchoff, MD**, Endocrinology, 2644 Jefferson  
Park Circle, Charlottesville VA 22903

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**Wayne D. Old, MD**, Internal Medicine, 201 Shirley Ave.,  
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**Sharon A. Pancoast, MD**, Internal Medicine, 806 West  
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**George P. Piros, MD**, Radiology, Box 167, RR 3, Char-  
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**James R. Poliquin, MD**, Surgery, PO Box 843, Norfolk  
VA 23501

**Susan M. Pollart, MD**, Family Practice, 1516 Broad Ave.,  
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**Moises E. Quinones, MD**, Internal Medicine, 2201-A  
Yellow Mountain Road, Roanoke VA 24014

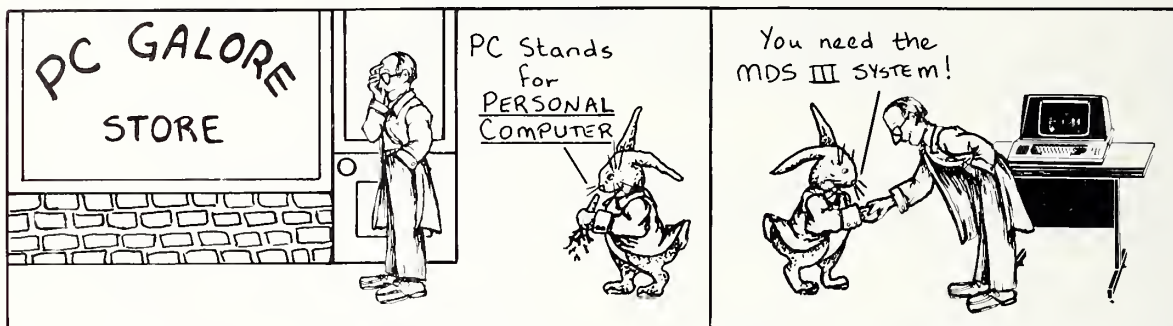
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## VIRGINIA PHYSICIANS AT THE GENERAL ASSEMBLY

**F**or the first time, Virginia physicians joined this year in a concerted effort to make themselves seen and heard at the General Assembly.

The campaign was set in motion by Dr. C. Barrie Cook, Medical Society of Virginia president, and Dr. Wyatt S. Beazley III, president of the Richmond Academy of Medicine.

Forty physicians from over the state converged on the capitol for a morning, or an afternoon, or a day at the legislature. Some came more than once.

Buttonholing delegates and sena-

---

*Dr. John M. Daniel III, Richmond, stands by at a subcommittee hearing on licensure of respiratory therapists. Seated on the far side of the table is Sen. Joseph V. Gartlan, Jr., (D-Alexandria). Half-hidden at the doorway is Jim Darden, aide to Del. Owen B. Pickett (D-Virginia Beach), who was conducting the hearing.*

---





tors, the visiting doctors asked questions, pondered answers, argued viewpoints, made mental notes for future reference. In committee hearings they witnessed the measured tempo of the democratic process, Virginia style.

Most had never seen their state legislature in action before.

Geographical propinquity swelled the Richmond contingent, but as the pictures on these pages illustrate, many MSV members made lengthy motor trips to get there.

Did they make a difference?

"Oh yes, a tremendous difference," said Melanie Rhoades, who as director of legislative affairs for the Richmond Academy bird-dogs the General Assembly.

"People from every other special interest group in the state are always there, pushing for what they want, but the doctors have always been strangely absent. 'Well, where are your doctors?' the legislators would say to me.

"This year the doctors were there, and the reaction of the legislators was wonderful, they really felt complimented by the doctors' visits."

Will Osburn, who lobbies for The Medical Society of Virginia, got the same reaction.

"The legislators were just amazed that the doctors would take the time to come down there," he said. "It really pleased them."

For how the visiting physicians reacted to the legislators, see Melanie Rhoades' account on page 270.

On page 273 is a roundup of this year's legislative action affecting Virginia physicians.

And to read why Dr. Robert L. Adeson thinks the new breast cancer/informed consent law can have healthy consequences for both physicians and patients, turn to page 274. The bill itself appears on page 277.

—A.G.

Photographs by Tim Wright

*Sitting in his office, Sen. Wiley F. Mitchell (R-Alexandria) emphasizes a point as he talks to Dr. Wallace E. Baker, Springfield.*



*The Speaker of the House, A. L. Philpott (D-Henry) makes vehement response to a question from Dr. Henry S. Campell, Martinsville, in Philpott's office.*



## SHOCKED AND AMAZED AT ASSEMBLY

**T**raining sessions for the physician volunteers well in advance of the General Assembly's opening kicked off the Richmond Academy of Medicine's 1984 program of physician visits to the legislature.

The training focussed on the issues to be introduced during the session and some do's and don't's to follow with the lawmakers. For instance, "DO be positive when discussing the issues; DON'T get angry with the legislators—at least not until you get away from the General Assembly Building."

Both the Richmond and the out-of-town doctors began their visits with briefings at either an 8 AM breakfast or noon-time luncheon. During this interval they had time to look over the Legislative Bulletin put together by Medical Society of Virginia staff, which describes the issues of particular interest to the medical profession and organized medicine's positions on them.

They could also look over the



*Dr. Hazel S. Konerding, Richmond, listens to Del. Thomas W. Moss.*

Virginia Legislative Directory, a booklet with pictures of all of Virginia's senators and delegates. Some of the visiting physicians were surprised to find childhood friends and classmates now serving in the Assembly.

At the General Assembly Building, the physicians met legislators, or their aides, and spoke to them on issues of particular concern. Then they attended committee or subcommittee hearings on bills relating to medicine. Some days a dozen doctors were moving about the General Assembly Building.

Most of the volunteers were shocked by the frenzied atmosphere—the constant moving about of legislators, aides and visitors as hearings are being conducted; the rush of traffic through the halls, with legislators intercepted every few feet by lobbyists pleading for this cause or that.

The visitors were amazed that so many meetings could go on at

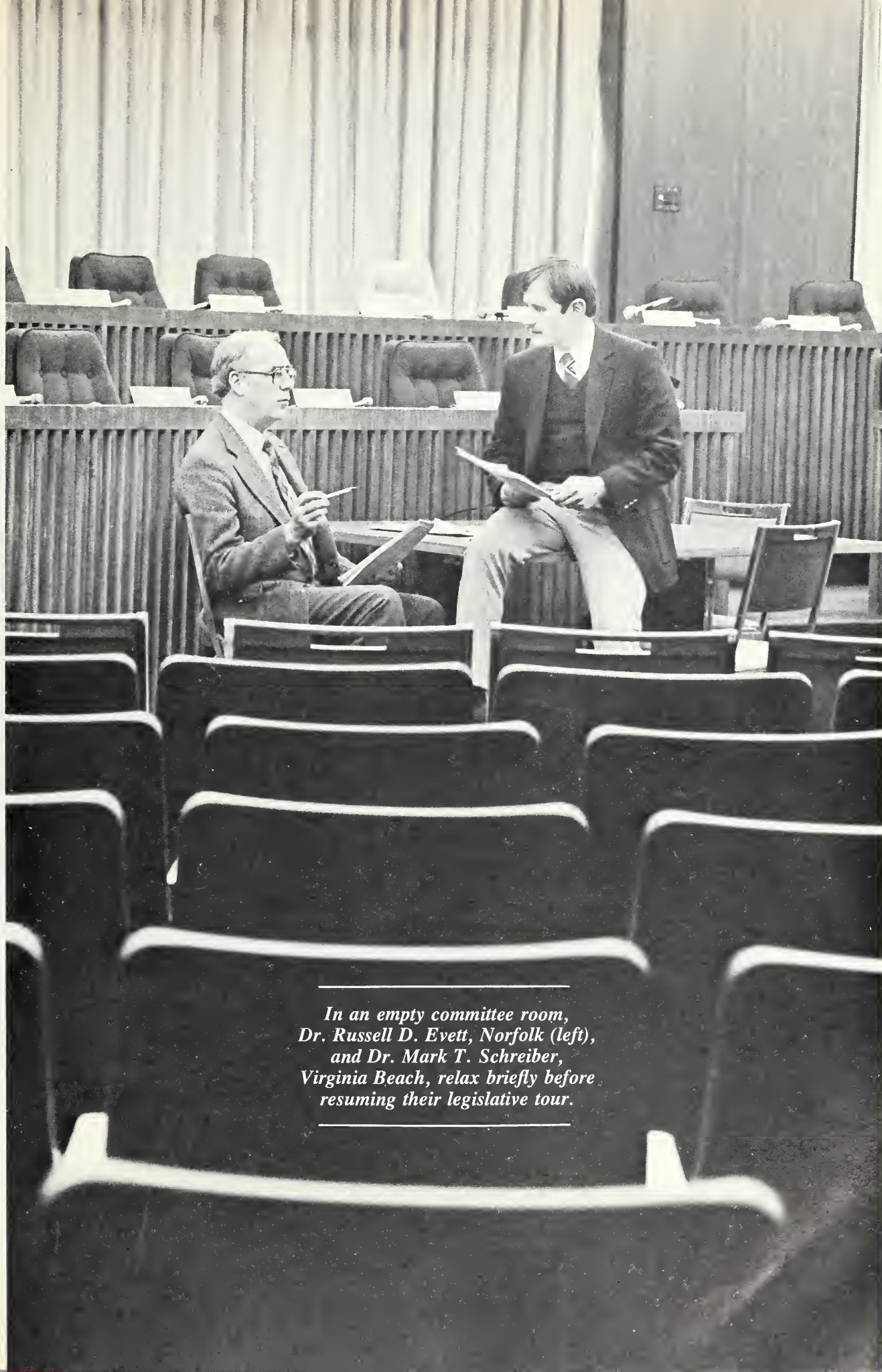
the same time and concerned that no single person could possibly know everything that went on every day. "Who's in charge of this madhouse?" one doctor asked in wonder.

From all the visiting physicians, there were expressions of satisfaction that they had taken the time to see and hear how laws are made in Virginia.

The Academy intends to continue its program at the 1985 session. In fact, Dr. Lawrence E. Blanchard III, chairman of the Legislative Committee, is planning to get members to visit delegates and senators well in advance of the 1985 session and register their views on matters relating to medicine. Also, the legislature set up a committee to study aspects of malpractice, and Academy members are to be assigned to monitor and contribute to those deliberations. And there are plans to involve more retired physicians in the 1985 effort.

—MELANIE RHOADES





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*In an empty committee room,  
Dr. Russell D. Evett, Norfolk (left),  
and Dr. Mark T. Schreiber,  
Virginia Beach, relax briefly before  
resuming their legislative tour.*

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*As the Senate Education and Health Committee prepares to open a hearing, three physicians from Richmond confer: from left, Dr. Henry H. Wilson, Jr., Dr. Donald G. Seitz, and Dr. Willys M. Monroe.*

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## THESE WERE THE BILLS IN '84

**H**ere summarized are actions emanating from the 1984 General Assembly that are of more than passing interest to Virginia physicians. The entries were selected for this roundup by Will Osburn, The Medical Society of Virginia's legislative liaison man, and Allen C. Goolsby III, the Society's counsel. Together with Sandra Kramer, an associate of Mr. Goolsby, they monitor the state legislature's every move.

### Malpractice

By passing a resolution sponsored by The Medical Society of Virginia, the General Assembly created a joint subcommittee to study Virginia's medical malpractice laws, including an evaluation of malpractice review panels. Four bills introduced at this year's session will be included in the study.

- HB-1011, to extend the \$1 million cap to the limits of the provider's insurance coverage, if that figure is larger;

- HB-664, to repeal the \$1 million cap;

- HB-668, to replace with a national standard Virginia's current

statewide standard of medical care;

- HB-669, to provide for the use of expert witnesses from anywhere in the United States.

The study committee is to consist of three legislators, two lawyers, one physician, and one hospital administrator.

The Speaker of the House is to appoint two of the legislators from the House Committee of Courts of Justice; the physician from The Medical Society of Virginia; and one of the two lawyers from the Virginia State Bar.

The Senate Committee on Privileges and Elections is to appoint the other lawyer from the Virginia Bar Association; the third legislator, this one from the Senate Committee of Courts of Justice, and the administrator, from the membership of the Virginia Hospital Association.

### Informed Consent/Breast Cancer

By passing SB-350, Virginia's legislators agreed to amend the Code of Virginia by stipulating that "before a physician operates on a patient for a tumor of the breast, the patient must execute a consent form that includes a section in which the patient designates one of the following options: 1) biopsy; 2) biopsy and surgery; 3) surgery.

### Mental Health

House Bill 4, which would completely revise the procedures for involuntary commitment to provide greater protection of patients' rights, was carried over to 1985.

Also carried over to 1985 was SB-218, relating to the disclosure of patient information by mental health professionals to third-party payers.

### Allied Health Professions

Senate and House bills relating to the licensure of respiratory therapists were carried over to the 1985 session.

*cont'd on page 276*

## CASTING A VOTE FOR CANCER CONSENT

**D**uring the 1982 session of the General Assembly, then-Del. Edythe C. Harrison introduced House Bill 406 relating to informed consent for treatment of breast cancer. After three versions of the bill were developed and discussed, the House version was "passed by indefinitely" by the Senate Committee on Education and Health.

The medical community, working with the American Cancer Society, Virginia Division, subsequently produced a brochure designed to educate women about alternative treatments available for breast cancer. [Editors' Note: The brochure was offered to Virginia physicians in VIRGINIA MEDICAL's November 1982 issue, which was devoted to the subject of breast cancer and informed consent.]

Many women remained seriously concerned about the issue however, and in response to their concerns, Sen. Richard L. Saslaw (D-Annadale) introduced Senate Joint Resolution No. 41, which established the Joint Subcommittee Studying Informed Consent

for Breast Cancer Treatment.

I served on that subcommittee.

At the public hearing that preceded our deliberative meetings, advocates of legislation made several different proposals, including 1) prohibiting the one-step (biopsy/mastectomy) procedure; requiring 2) that all patients receive educational brochures or 3) that all patients be informed of all alternative treatments for breast cancer; 4) legislating a surgical consent form.

The subcommittee's subsequent meetings culminated in the recommendation that a specific consent form be required prior to breast biopsy. As proposed, this form makes it absolutely clear what is being done. Surely, with this permit, nobody could claim that a mastectomy had been performed without expressly authorized consent.

It does not, however, satisfy all the wishes of those who have breast cancer. Massachusetts has a law, for instance, stipulating in part that "... a patient suffering from any form of breast cancer [is entitled] to complete information

on all alternate treatments which are medically viable."

Nor does the consent-form legislation insist that an educational brochure on breast cancer be distributed to all patients, as is now the law in California.

There are valid reasons why neither of these concepts were included in the subcommittee's recommendations. First, the Massachusetts law uses the words "complete," "all," and "medically viable"—terms that are open to such a variety of interpretations as to make them unrealistic in everyday doctor-patient discussion.

As for a mandated brochure, such as California's, there are many troublesome questions. For example, how will the brochure's information be kept up to date and by whom? What about the patient who exercises her right not to know and chooses not to read the brochure—should the physician treat her?

Certainly educational brochures should be used extensively when indicated, the subcommittee was agreed, but not under the



mandate of a legislative code.

The American Cancer Society, Virginia Division, and The Medical Society of Virginia should keep the brochure up-to-date and should continue to distribute it throughout the state. The state medical and surgical societies should ask their members for full cooperation not simply in distributing the brochure but in educating their patients to the brochure's information.

After consideration of the alternatives, the subcommittee selected the consent form as its legislative proposal.

**M**y initial instinct was that legislation was not the answer to the concerns expressed by the subcommittee. The risks associated with legislating medical treatment of one disease are not insubstantial. I am especially concerned about starting a statutory laundry list of special requirements for different medical problems.

Yet I cast my vote on the subcommittee in favor of accepting the legislation if it was to be limited to a required consent form.

The women who appeared before us had one fundamental concern, namely, being subjected to a mastectomy without their knowledge and consent. I doubt this possibility exists today, but many women do not share my belief. Whether it truly exists or is perceived to exist is moot; women are requesting strong assurance that no mastectomy be done without clearly expressed written consent.

Since I believe it is of utmost importance that physicians, especially surgeons like myself, make clear that we are sensitive to this issue, I supported the recommen-



*Signing the bill:  
from left, Sen. Saslaw, Dr. Adeson, Gov. Robb.  
To see the bill itself,  
turn the page.*

dation of the subcommittee that the General Assembly enact legislation that states that a patient will not be deemed to have consented to a mastectomy unless a consent form has been executed that expressly authorizes a mastectomy. The law was passed by both House and Senate and signed into law by Gov. Charles S. Robb. It becomes effective July 1. [The bill in its entirety is on page 277.]

I think the subcommittee came up with a solution to the breast cancer problem which will satisfy everyone's wishes and not put anyone in an undesirable position.

I believe the impact of this legislation designed to improve the mechanism of consent will extend beyond the form itself and will increase satisfactory patient-doctor communication.

We must acknowledge that over the past several years the medical establishment has not communicated as well with the community and with our patients

as we should. Patients' trust in us has wavered, and because of the ever-present malpractice problem, physicians are increasingly uneasy. Patients and doctors are warier and often look upon each other as adversaries.

Therefore, I think the medical community should look upon this kind of legislation as an opportunity to demonstrate that we want to close the gap between the community and us, that we care about our patients, and that we can work with the legislature when necessary to help solve common problems.

I believe that once Virginia doctors realize that this legislation is providing a service, that it doesn't encroach on the way they want to practice or tell them how to practice, they will find it acceptable.

—ROBERT L. ADESON, MD

Dr. Adeson is a general surgeon practicing at 5001 Seminary Road, Alexandria VA 22311.

Killed in the Senate Education and Health Committee was a House bill redefining the practice of physical therapy to permit independent evaluations. It probably will be reintroduced in 1985.

The occupational therapists were expected to sponsor a licensure bill, but it did not materialize.

A resolution to study the need for licensure or certification of athletic trainers died in committee.

#### **Certificate of Need**

Two bills from the Senate and one from the House had to do with certificate of need exemptions.

One asked for exemption for outpatient, or ambulatory, surgery; renal dialysis; radiation therapy; and CT scanning centers. It was carried over to 1985.

The other sought to exempt all home health agencies. It was killed.

A third bill would have repealed the certificate of need laws. It was carried over to 1985.

#### **Pharmacy**

Four bills of note relating to pharmacy were passed.

One requires every physician who works in a free-standing emergency center to print name, address, telephone number, and federal controlled substance registration number on the center's prescription blank when the physician issues it.

A second stipulates that any insulin preparation must be dispensed by or under the supervision of a licensed pharmacist.

Methaqualone was placed on Schedule I with another bill.

Sample drugs given to physicians by detail men were the subject of the fourth bill, specifically, drugs in Schedules II through V. Such drugs may not be left at the physician's office unless the physician has requested the drugs or execut-

ed a written confirmation of their receipt.

#### **Assignment**

Passed was HB-780, which provides that a patient cannot be denied the right to assign benefits under a prepaid health plan to a non-participating provider or hospital, except when the benefit is 80% of covered charges or greater.

#### **Drunk Driving**

Much media attention surrounded a number of bills concerned with drunk driving.

One bill that passed stipulates that if the operator of a motor car is found to have a blood alcohol content of more than 0.15%, that person is deemed to have committed the crime of drunk driving.

A bill to raise the legal age for drinking beer to 21 years was defeated.

#### **Abortion**

Killed in committee was a bill that would have restricted abortion by requiring specific disclosures by the attending physician to obtain "informed consent" and notification of the families of minors seeking abortions.

#### **Miscellaneous**

Not-for-profit health maintenance organizations would have been classified as charitable organizations and exempted from property taxes if HB-732 had passed, but it was killed in committee.

A bill seeking licensure for anyone practicing social work was carried over to the 1985 session.

Also carried over was HB-896 having to do with reporting of results of medical malpractice suits. Plaintiff's attorney and the malpractice insurer must report claims to the Commissioner of Insurance as they are settled or go to trial, the bill specifies, and from the submitted information the Commissioner is to prepare an annual report.

The legislators removed from the Code of Virginia the long-standing requirement for a premarital blood test for syphilis.

A Senate bill relating to the liability of spouses for medical care was passed. Each spouse shall be jointly liable for all emergency medical care furnished to the other spouse by a physician licensed to practice medicine in Virginia or by a hospital located in Virginia, says the bill. Included in the liability is followup inpatient care during the initial emergency admission. The bill stipulates that the spouses must be living together at the time the care is delivered.

Passage of SB-142 paves the way for a central registry of persons who have sustained head injuries that may result in permanent disability.

Subject to enactment of federal funding legislation, Medicaid eligibility for prenatal care for first-time pregnancies is mandated by SB-200, which passed.

Epinephrine was the subject of a Senate bill that 1) repealed the statute relating to administration of epinephrine in emergency situations and 2) excluded from liability a person who, in an emergency situation, administers epinephrine to a person for whom an insect-sting kit has been prescribed.

Clarification of the Natural Death Act passed in 1983 was promised with passage of a bill from the Senate. It states that the administration of medication and the performance of any medical procedure for the purpose of providing comfort care of alleviating pain are not to be considered "life-prolonging" procedures.

Passage of HB-231 makes it a criminal offense to sell body parts, excepting hair, blood and other self-replicating body fluids. The House and Senate also passed a bill permitting the harvesting of temporal bones.



1984 SESSION

CHAPTER

83

REENROLLED

*An Act to amend the Code of Virginia by adding a section numbered 54-325.2:2, relating to informed consent for treatment of breast tumor.*

[S 350]

Approved MAR 8 1984

Be it enacted by the General Assembly of Virginia:

1. That the Code of Virginia is amended by adding a section numbered 54-325.2:2 as follows:

§ 54-325.2:2. *Informed consent for treatment of breast tumor; paragraphs required in form.—Before a physician operates on a patient for a tumor of the breast, a consent form shall have been executed which includes the following:*

‘‘CONSENT FOR TREATMENT OF BREAST TUMOR’’

Sign option (a) or option (b), or option (a) and option (b).

(a).....Breast Biopsy

Side (right or left)

.....  
Patient's or other authorized person's Signature

(b) If it is determined that I have a malignant tumor in my breast or other breast abnormality requiring surgery, then I authorize Dr. ....  
..... to perform such operations or procedures, including breast removal, which are deemed necessary.

Procedure:

.....  
Patient's or other authorized person's Signature

*Richard J. Dam*

President of the Senate

*A. Z. Philpott*

Speaker of the House of Delegates

Approved:

*Charles S. Robb*

Governor

# Survey of Burn Injuries in Virginia

Richard F. Edlich, MD, Ernst O. Attinger, MD, and  
Antharvedi Anné, PhD, *Charlottesville, Virginia*;  
Willcox Ruffin, Jr., MD, *Norfolk, Virginia*; and  
Boyd W. Haynes, MD, *Richmond, Virginia*

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In cooperation with 89 Virginia hospitals, the authors studied data on the incidence and epidemiology of both minor and serious burn injuries. Significantly, they found the statewide emergency referral system functioning successfully in routing the severely burned to Virginia's four burn treatment centers.

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**B**URNS cause severe injuries that result in significant physical and psychological trauma to the patient. Even though it is an important public health problem, the incidence of these injuries is not known. Most studies have been confined to a limited number of hospitals and do not convincingly provide incidence rates in representative populations. Without such data, the true dimensions of burn injuries cannot be appreciated.

This absence of data on the incidence of burn injury caused the United States Congress to enact a

From the Department of Plastic Surgery (Dr. Edlich) and the Department of Biomedical Engineering (Dr. Attinger and Dr. Anné), University of Virginia School of Medicine; Norfolk General Hospital (Dr. Ruffin); and the Department of Surgery, Medical College of Virginia (Dr. Haynes). Address correspondence to Dr. Edlich at Box 332, University of Virginia Medical Center, Charlottesville VA 22908.

Submitted 6-28-83.

National Burn Injury Program, under the Public Health Services Act, Section 1221, as implemented by Health Services Administration. The legislative purposes of this program were to document the incidence and epidemiology of burn injury, to describe the current burn delivery system and to collect data and information on the process of burn care. Since 89% of the hospitals in the Commonwealth of Virginia agreed to participate in the study, we have prepared a summary of the results of this project in grateful recognition of their commitment to this most important study.

## MATERIALS AND METHODS

The Department of Health, Education and Welfare selected six regional sites to implement the investigation, one being the Commonwealth of Virginia. The University of Virginia supervised the collection of data in Virginia by three data retrieval



teams located in Norfolk, Roanoke and Charlottesville. The Tidewater Emergency Medical Services Council, Incorporated, and Western Virginia Emergency Medical Services Council provided administrative supervision for the data collection staffs in Norfolk and Roanoke, respectively. A portion of this comprehensive project was devoted to collecting data for a 21-month period for burn patients whose injuries were judged to be serious enough to warrant hospitalization (serious burns). During 17 of these 21 months, data was also collected from emergency departments on burn patients whose injuries were not considered serious enough to justify hospitalization (minor burns). Patients who received care at home or in the office of a physician or who had smoke inhalation without a burn were not included as part of the study. The strategy for data collection, and the quality control and monitoring of collected information has been previously reported.<sup>1</sup>

## RESULTS

### Incidence of Burn Injury

In this study, 1552 burn patients sustained serious burn injuries over a 21-month interval that required treatment in a hospital; 79 of these patients died. The annual incidence of serious burn injuries was 902 patients. On the basis of the Census Bureau Data for Virginia, it is estimated that 20 Virginians per 100,000 population sustained a serious burn injury requiring treatment in a hospital setting each year.

The incidence of minor burn injuries in Virginia was more than ten times greater than that of serious burn injury. During the 17-month interval in which data was collected from emergency departments, 17,384 patients with minor burn injuries were recorded. The annual incidence of these minor burn injuries was 11,662, occurring in approximately 250 Virginians per 100,000 population per year.

People of all ages were susceptible to serious and minor burn injuries. A higher incidence of minor (62.0%) and serious (72.1%) burns occurred in men than in women, and a greater incidence of minor (73.8%) and serious (70.6%) burn injuries occurred in the white population than in the non-white population. The highest incidence (32.4%) of minor burn injuries occurred in the young adult, ages 20-29. Men in this age group also had the highest incidence of serious burns (22.9%). For women, the highest incidence (28.6%) of serious burns was evident in the age group 0-9 years.

An individual's risk for burn injury was computed

by comparing the age, race and sex of the burn population in Virginia to that of the population in Virginia described in the census data. The risk factors (RF) have been calculated so that a value of one is considered average, while values above one indicate a greater risk of burn injury than the general population.

The patient's sex and race were important determinants of risk for serious and minor injuries. Men (RF 1.45,  $p < .05$ ) had a significantly greater risk for serious burn injury than the average population, while women (RF 0.55,  $p < .05$ ) had a lower than average risk. Similarly, men (RF 1.26,  $p < 0.01$ ) had a significantly greater risk for minor burn injury than the average population, while women (RF 0.75,  $p < 0.01$ ) had a lower than average risk. The non-white population's risk for minor (RF 1.38,  $p < 0.01$ ) and serious (RF 1.53,  $p < 0.05$ ) burn injuries was significantly greater than the white population's risk for minor (RF 0.91,  $p < 0.01$ ) and serious (RF 0.87,  $p < 0.05$ ) burn injuries. The non-white male population exhibited the highest risk for serious (RF 2.12,  $p < 0.01$ ) and minor (RF 1.69,  $p < 0.01$ ) burn injuries, followed by the white male (serious RF 1.30,  $p < 0.05$ ; minor RF 1.16,  $p < 0.01$ ), non-white female (serious RF 0.98,  $p = \text{NS}$ ; minor RF 1.08,  $p < 0.01$ ) and then white female (serious RF 0.45,  $p < 0.05$ ; minor RF 0.67,  $p < 0.01$ ).

The age of the population had considerable influence on their risk for serious and minor burn injuries (Fig. 1). The risk for minor burn injury was greatest during two periods of life, age one year and ages 20-29 years. Outside these peaks, the age-specific risk factors for minor burn injury decreased progressively until designated age groups displayed a reduced risk for burn injury. The risk for minor burn injury was highest in one-year-olds (RF 2.52,

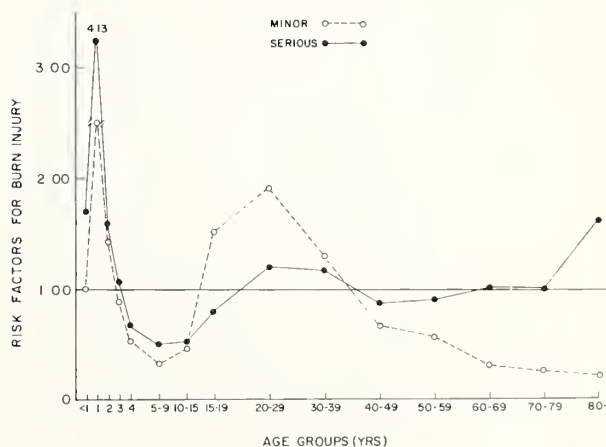


Fig. 1. Age-specific risk factors for burns.

$p < 0.01$ ). The risk for minor burn injury then decreased progressively until age 4 years (RF 0.5,  $p < 0.01$ ), when the risk was significantly below that of the average population. This reduced risk persisted until age 15 years (RF 1.57,  $p < 0.01$ ), at which time the risk for burn injury was above that of the average population. This enhanced risk of burn injury became even greater at ages 20-29 years (RF 1.95,  $p < 0.01$ ) and then gradually decreased until age 40 years (RF 0.69,  $p < 0.01$ ), at which time it was again below that of the normal population. This decreased risk for burn injury was also evident in individuals who were older than 40 years.

The risk for serious burn injuries was greatest during three periods of life, age one year, ages 20-29 years, and ages 80 years and older. Outside these peaks, the age-specific risks for serious burn injury decreased progressively until the designated age groups displayed a reduced risk for burn injury. The risk for serious burn injuries was highest in one-year-olds (RF 4.13,  $p < 0.01$ ), then decreased progressively to age 4 (RF 0.69,  $p < 0.01$ ), when the risk was significantly below that of the normal population. This reduced risk for serious burn injury persisted until ages 20-29 years (RF 1.22,  $p < 0.01$ ), at which time the risk for serious burn injury was above that for the average population. The enhanced risk of serious burn injury persisted in age group 30-39 years (RF 1.19,  $p < 0.01$ ) and then decreased progressively at ages 40-79 to a level that did not differ significantly from that of the average population. For patients 80 years and older, an increased risk for serious burn injury (RF 1.65,  $p < 0.01$ ) was readily apparent.

### Mechanism of Burn Injury

The mechanism of minor burn injuries varied considerably. A large number of different heat sources caused a significant number of burn injuries. There were five major causes of burns (liquid scald, hot surfaces, chemical, radiation, grease scald), each of which accounted for more than 10% of the cases. The single most important cause of minor burn injury was scald by hot water, which accounted for 18.9% of the cases. Contact with a hot surface was the second most common cause (17.0%). In contrast, the major causes of severe burn injury were restricted to two etiologies, flame and scald liquid, for the population of hospitalized burn patients. These two heat sources accounted for the majority (60.2% of 1552 serious burn injuries. Flame accounted for the largest number of serious burn injuries (36.7%), while liquid scald was second (23.5%).

When the mechanism of minor and serious burn injury was correlated with the patient's age, there were large differences in the age-specific incidence rates. This correlation between the patient's age and the mechanism of burn injury is graphically displayed in Figures 2 and 3. While scald liquid burns accounted for a significant number of minor and serious burn injuries in all age groups, it was the major cause of serious burn injury in children 5 years old and younger (Fig. 2). The risk for serious scald (liquid) burns was highest in one-year-olds, accounting for 67.3% of the serious burn injuries. After the age of 5, flame burn was the major cause of serious burn injuries, accounting for nearly one-third or more of the cases (Fig. 3).

The mechanism of burn injury had an important influence on its ultimate outcome (Fig. 4). When the severity of burn injury was measured by the abbreviated burn severity index, the frequency of burn injury caused by flame burn increased as the patient's estimated probability of survival decreased. Of the burn injuries that were severe enough to cause the death, 82.3% were due to flame burn.

### HOSPITAL RESOURCES FOR BURN CARE

The resources of hospitals for delivering specialized burn care was determined using categorization guidelines prepared by the American Burn Association.<sup>2</sup> This organization outlined specific optimal criteria for burn centers with special expertise in burn care. The most significant ingredient necessary for optimal care of the burn patients was commitment, both by personnel and the institution. Utilizing the specific optimal criteria, a questionnaire was developed and sent to all hospitals in the Commonwealth of Virginia to assess their ability to deliver specialized burn care. This voluntary as-

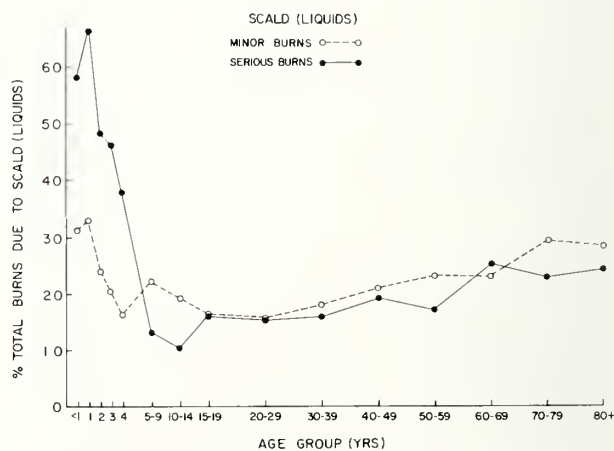


Fig. 2. Age-specific burn incidence due to liquid scald.



assessment of the hospital resources was confirmed by a visit by our staff to each participating hospital. The resources of four of the participating hospitals were sufficient to permit their designation as burn centers with special expertise in burn care. These four burn specialty treatment centers cared for 39.4% of the total burn population. The remaining hospitals, which did not exhibit this level of specialty care capability, are referred to in this study as community hospitals.

### Regional Referral System

If a regional referral system was judged to be successful in altering the distribution of burn patients, the specialty burn treatment facility should be caring for the more seriously burned patient, while hospitals without special expertise in burn care should be involved in treating the less seriously burned patient. The severity of burn injuries of patients treated at each hospital setting was measured by the percentage of body surface area burned, abbreviated burn severity index, and mortality.<sup>3</sup> Seventy-three of the 74 burn patients with second- and third-degree burns comprising more than 41% of the body surface area were cared for in the specialty burn centers. Only one patient with this magnitude of burn injury was cared for in a community hospital. When the abbreviated burn severity index was used to assess the seriousness of burn injury of the survivors, patients whose chance for survival was estimated to be 40% or less were treated primarily in the specialty burn treatment facilities. Of the 53 survivors with this severity of burn injury, 48 were cared for in burn centers. Of the 1552 patients, 79 succumbed as a result of the injury; 70 of these patients died in regional burn centers.

Since the more seriously burned patients display

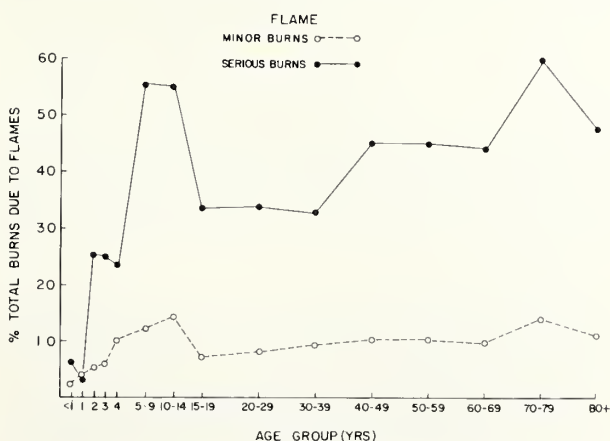


Fig. 3. Age-specific incidence of burns due to flame.

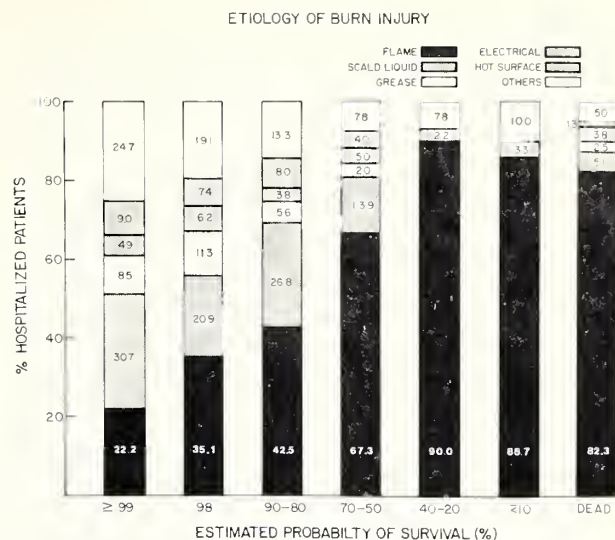


Fig. 4. Incidence of burns due to flame was proportional to severity of injury.

a higher risk of developing complications than the less seriously burned, it would be anticipated the hospitals treating the more seriously burned patients would encounter a higher incidence of complications than those treatment facilities caring for patients with the minor burn injuries. When the complications experienced by the burn patient were correlated with the hospital treatment facility, the incidence of respiratory, neurologic and psychiatric complications was significantly higher in patients treated in the burn centers than those cared for in hospital settings without special expertise in burn care. The exception to this rule was patients with delirium tremens whose distribution within the hospital treatment settings did not differ significantly from that of the total burn population.

### Process of Care

Since the more severely burned patients were treated in specialty burn centers, it was expected that this would influence the process of burn care from the time of arrival in the hospital through rehabilitation. Treatment of the more seriously burned patient should involve more comprehensive and specialized care than that required to manage effectively the less seriously burned patient. Respiratory support, hyperalimentation, hemodialysis, enzymatic debridement of wounds, and blood transfusions were performed to a greater degree in the regional burn center than in hospitals without special expertise in burn care. The only treatments that were performed with similar frequency in a burn center and hospitals without special expertise in burn care were topical antibacterial therapy and the

application of gauze bandages to the burn wound. The frequency with which burn patients required rehabilitation services was significantly greater in the burn center than that in the community hospital setting.

## DISCUSSION

There is an increased recognition among physicians, educators and legislators that the incidence of burn injury in our country is a national disgrace. One of the goals of the National Burn Injury Program was to document the magnitude of the problem. Historically, the quantification of the incidence of a disease in a population has been an essential step in the control of the disease. On the basis of this study, it is estimated that each year 20 Virginians per 100,000 population sustain serious burn injuries requiring treatment in hospital settings. The incidence of minor burn injuries in the Commonwealth of Virginia was more than ten times greater than that of serious burn injuries. The etiology and frequency of minor and serious burn injuries have been found to differ according to the age, race and sex of the patient. Examination of our data revealed distinct patterns for the magnitude of age-specific incidence of minor and serious burn injuries for the designated race and sex. This distribution of the age-specific incidence rates indicates that minor and serious burn injuries occur non-randomly in specific etiologic categories. These findings can now be used as a starting point for identifying the high-risk populations in Virginia that can benefit from targeted educational programs.

In the future, we plan to survey this high-risk population in Virginia to determine if there are common misconceptions and knowledge deficiencies about the epidemiology of burn injury. A variety of educational strategies have been developed to alter their behavior. These include public information campaigns making extensive use of mass media, curricula for elementary and secondary schools, and programs initiated by adults in community organizations. The effect of these strategies upon the understanding by the population of its message as well as its behavior must be examined to evaluate the impact of these educational programs.

It is generally agreed that an echelon system of care for patients with life-threatening burn injuries should be developed on a regional basis. This system's approach to burn care is based on the assumption that patients are served more effectively and efficiently when the activities of the health care providers are coordinated. Regional planning

and development of specialty burn treatment centers has the potential for limiting unnecessary spending through concentration of capital and personnel, improving the quality of care by maintaining the skills of burn care teams, and establishing suitable mechanisms for transportation and referral of patients through coordination with emergency medical service systems.

To improve patient care, cost effectiveness and resource utilization, the Emergency Medical Services of the Commonwealth of Virginia have attempted to regionalize burn care services by identifying specialized burn treatment centers for care of the seriously ill burn patient. Special guidelines that identified the seriously injured burn patients that should be transferred to the regional burn centers were prepared and distributed in poster form to all hospitals in the state.<sup>4</sup> The performance of this system has been evaluated in this study by identifying the distribution of burn patients in the hospitals of the Commonwealth of Virginia. The high proportion of seriously burned patients being treated in the burn specialty care centers suggests that the regional burn referral system appropriately moved them to the specialized burn treatment center. Moreover, the specialty care burn centers had sufficient number of beds to care for patients whose burn injury was judged to be life threatening. Consequently, hospitals without special expertise in burn care were not faced with the dilemma of caring for critically ill burn patients without adequate resources. Implicit in this finding is that additional regional burn centers are not presently needed in the Commonwealth of Virginia. Our strategy for evaluating the process of the burn care has obvious implications for other regional referral systems for high-risk patients (i.e., spinal cord injury, head injury, high-risk infant, etc.) and should serve as a model for assessing their performance.

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# Current Concepts of Cardiopulmonary Resuscitation

Barry E. Yarbrough, MD, *Richmond, Virginia*

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Survival of the cardiac arrest patient depends on prompt cardiopulmonary resuscitation to provide cerebral blood flow, followed by restoration of coronary flow. For prolonged arrest, open-chest massage may be indicated. Drug therapy is controversial; recent evidence suggests that calcium chloride may be deleterious, but slow channel calcium blockers are probably useful.

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ONE of the earliest descriptions of mouth-to-mouth ventilation dates to a 1775 meeting of England's Royal Humane Society for the Apparently Dead<sup>1</sup>:

*The most efficacious are to blow with force into the lungs, by applying the mouth to that part of the patient, closing the nostrils with one hand, and gently expelling the air again by pressing the chest with the other. . .the medium of a handkerchief or cloth may used to render the operation less indelicate. . .Expand the lungs as soon as possible. . .much depends on this. . .*

Two hundred years later the concept of mouth-to-mouth resuscitation was rediscovered and popularized.<sup>2</sup> With the development by Kouwenhoven of a human defibrillator and first successful defibrilla-

tion of a human heart by Dr. Claude Beck in 1947,<sup>3</sup> the modern age of cardiopulmonary resuscitation (CPR) had begun.

In response to the growing need to educate physicians and other health care workers in the principles of CPR, a Lincoln, Nebraska, physician, Dr. Stephen Carveth, developed the Advanced Cardiac Life Support (ACLS) course and presented it under the sponsorship of the American Medical Association in 1974. The course was further developed by Dr. Arnold Sladen of the American Heart Association (AHA) and has been presented by the AHA since 1975. The course has been widely accepted and a growing number of hospitals require their emergency medical personnel to be ACLS-certified. Although the information in the ACLS course is presented in doctrinaire fashion, current research work questions some of the basic tenets taught in ACLS.

## Mechanism of Blood Flow During CPR

After 1960, when Kouwenhoven's classic paper describing the efficacy of closed-chest cardiac mas-

From the Department of Medicine, Medical College of Virginia/Virginia Commonwealth University, Richmond. Dr. Yarbrough is now practicing at St. Joseph's Hospital in Towson, Maryland.; address correspondence to him at 1211 Stevenson Lane, Towson MD 21204.

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sage was published in JAMA<sup>4</sup>, it was generally believed that blood flow during CPR resulted from compression of the ventricles between the sternum and spine. This view was not challenged in spite of reports of findings not consistent with this concept. In 1962 Weale and Rothwell-Jackson<sup>5</sup> noted that measured pressures in the iliac arteries and veins of animals were almost equal during closed-chest massage and questioned the efficacy of the new techniques. In 1964 MacKenzie<sup>6</sup> found nearly equal pressures in the right atrium and aorta of patients during external chest compression, suggesting that compression of the heart was not the mechanism of blood flow. Further evidence against the cardiac compression model was published by Werner et al<sup>7</sup> in 1981; they demonstrated, using the echocardiogram, that the mitral valve remains widely patent throughout the compression and relaxation phases of external chest compression.

These findings are best explained by the "thoracic pump" mechanism of blood flow during CPR (Fig. 1). This mechanism suggests that blood flows during closed-chest compression due to increased intrathoracic pressure, rather than compression of the heart, and that the heart acts as a passive conduit during CPR. Blood flow to the head and upper body is the result of internal jugular and other venous values not present in the lower extremities which prevent backflow of blood during the relaxation phase and produce a pressure gradient. Therefore, medications injected into the lower body veins, such as the femoral system, would not be expected to enter the central circulation rapidly, and cannulation of these veins during CPR does not seem advisable.

The primary objective of CPR is to perfuse the central nervous system while attempting to restore effective cardiac contractions. If carotid artery flow rates are dependent upon intrathoracic pressure, then any maneuver which would increase intrathoracic pressure during the compression phase should enhance cerebral blood flow and, hopefully, improve survival. One way of doing this is to ventilate the patient during the compression phase of CPR rather than during the relaxation phase as taught by the AHA. Chandra and his associates<sup>8</sup> have demonstrated increased arterial systolic pressure and carotid blood flow in animal studies utilizing simultaneous ventilation and chest compression.

During CPR the diaphragms are driven forward into the abdomen, resulting in transmission of some of the force of external compression to the abdomen. If the abdomen were bound to prevent this transmission of pressure, then perfusion of the

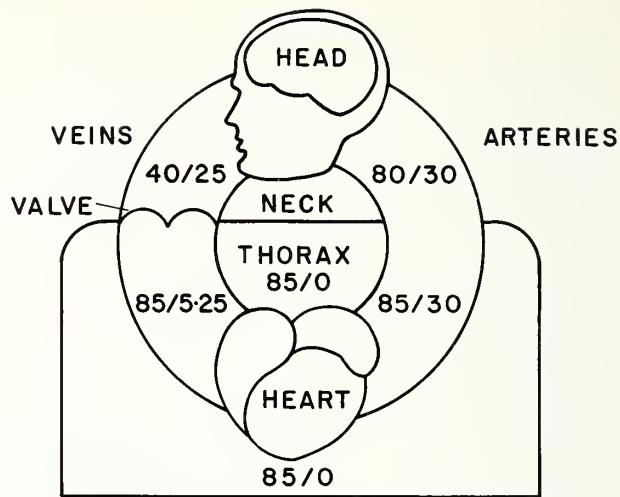


Fig. 1. Mechanism of blood flow during CPR.

brain should improve; recent studies<sup>9</sup> have shown that abdominal binding increases carotid artery flow rates.

If simultaneous ventilation and chest compression and abdominal binding increase carotid arterial flow and arterial systolic pressure, what would be the effect of combining these two techniques? Recently, Ewy and associates<sup>10</sup> attempted to answer this question by comparing results of resuscitation in a dog model employing simultaneous ventilation/chest compression and abdominal binding ("new CPR") versus conventional CPR. To their surprise, five of the six animals treated conventionally were resuscitated, but none of the six dogs treated with the new CPR could be resuscitated. A review of their hemodynamic data showed that the predominant difference between the two groups was a substantially higher aortic diastolic blood pressure in the survivors. Since coronary blood flow is a result of a gradient between the aortic diastolic and right atrial pressures, maneuvers such as abdominal binding, which decrease this gradient, may result in decreased coronary blood flow, leading to inability to restore effective spontaneous cardiac contractions. Thus maneuvers which increase carotid artery blood flow during CPR may not improve survival since maintenance of the arterial diastolic-right atrial pressure gradient is important to survival.

Studies such as these have led to a renewed interest in open-chest cardiac massage. Data from the Seattle Heart Watch<sup>17</sup> show that when CPR is begun within four minutes after the onset of ventricular fibrillation and arrival of ACLS is within eight minutes, the survival rate is 43%. Survival rate is only 10% or less, however, if ACLS is delayed 16



minutes or longer. Survival using closed chest massage with prolonged arrest is unusual, probably because conventional CPR, while perfusing the brain adequately, does not produce necessary coronary perfusion. Open-chest cardiac massage produces excellent coronary perfusion and therefore appears indicated for treatment of prolonged cardiac arrest. Treatment must be aimed at both the brain and the heart for a successful outcome.

## THErapy DURING CPR

### Epinephrine

In the early 1900s, an American surgeon, George Washington Crile, demonstrated that dogs could be successfully resuscitated using adrenalin, rhythmic chest pressure and artificial ventilation.<sup>11</sup> Epinephrine remains an important drug in the management of cardiopulmonary arrest. Since epinephrine has both alpha and beta effects, there has been controversy as to which of these actions is responsible for the beneficial effect of epinephrine during cardiopulmonary arrest. Otto and associates<sup>12</sup> utilized a model in which dogs were pretreated with an alpha (phenoxybenzamine) and a beta (propranolol) adrenergic blocker, or both, prior to cardiac arrest, and compared survival when the animals were treated with epinephrine and CPR. None of the animals receiving phenoxybenzamine alone or in combination with propranolol were successfully resuscitated, while six of the eight animals receiving propranolol and seven of eight animals receiving no pretreatment were successfully resuscitated. They concluded that the alpha adrenergic effects of epinephrine were responsible for the beneficial effects of epinephrine during CPR. Other alpha adrenergic agents including phenylephrine, metaraminol and methoxamine have been tested and recommended as equally effective alternatives.<sup>13</sup>

### Bicarbonate

The original rationale for routine use of bicarbonate during CPR was based on reports that epinephrine was less effective in the presence of severe acidosis (pH 6.76).<sup>14</sup> Subsequent studies have failed to support this conclusion<sup>15</sup>, yet bicarbonate is routinely administered in most resuscitation attempts. The routine use of bicarbonate has never been shown to improve survival during CPR. In most patients, initial respiratory alkalosis is seen within the first five minutes of the resuscitation attempt if the subject is ventilated forcefully every fifth chest compression. After five minutes a metabolic acidosis ensues to a mean value of pH 7.2 at 30

minutes in experimental animals.<sup>16</sup> This is a mean value; some subjects maintain normal or alkalotic pH.

Hazards of excessive bicarbonate administration include volume overload, hyperosmolarity, and increased affinity of hemoglobin for oxygen, which reduces tissue oxygenation. The American Heart Association recommends that bicarbonate administration should be guided by arterial blood gas determinations whenever possible.<sup>17</sup> In cases of arrest with rapid restoration of circulation, bicarbonate is usually not necessary. Frequently the pH may be controlled adequately by hyperventilating the patient. This technique also lowers intracerebral pressure and avoids possible untoward effects of bicarbonate, including possible "overshoot" alkalosis if resuscitation is successful and the excess lactic acid is converted back to bicarbonate.

### Calcium Chloride

The American Heart Association recommends the use of 5 ml of 10% calcium chloride every ten minutes in cases of asystole or electromechanical dissociation (EMD).<sup>17</sup> Only anecdotal evidence from the 1950s is offered as rationale for this recommendation.<sup>18</sup> Recent studies suggest that the use of calcium chloride may be harmful. Dembo<sup>19</sup> has shown that when given by AHA standards, serum calcium concentrations of 12.9 to 18.2 mg/dl (mean 15.3 mg/dl) were produced. A Milwaukee group retrospectively examined the results of CPR due to asystole and EMD and found that patients who did not receive calcium fared significantly better.<sup>20</sup> In that series, 8% of asystolic patients and 16% of patients in EMD were successfully resuscitated using calcium; without calcium, 33% of asystolic patients and 44% of patients in EMD were successfully resuscitated.

There has been considerable interest of late in the use of slow channel calcium blockers during CPR. White et al<sup>21</sup> demonstrated that post-arrest increases in cerebral vascular resistance could be successfully blocked by administration of flunarazine, a slow channel calcium blocker. Calcium ion entry into neuronal cells has been implicated in many cellular reactions producing neuronal death, including vasospasm, mitochondrial uncoupling, membrane degeneration and production of cytotoxic compounds.<sup>22</sup> The possible benefits of this class of drugs in resuscitation is one of the most promising recent developments in CPR. It seems unwise, therefore, to administer calcium chloride, a drug of unproven efficacy, when calcium ion entry into cells may be deleterious.

over

## Atropine

Atropine is the prototype anticholinergic drug and has been widely used as a cardiac accelerant. Recently Criley et al<sup>23</sup> have suggested that it may be a useful drug in asystole. They report eight cases of asystolic cardiac arrest in which an organized rhythm was obtained after administration of atropine. Three of these patients survived; this is a much higher rate of survival than the dismal outcome usually observed. However, the surviving patients arrested either in the catheterization lab or ICU and received immediate treatment; these results can not be transposed to prehospital asystolic arrest. While increased vagal tone etiology of brady-asystolic rhythms caused by anesthesia, intubation, vascular manipulations and certain forms of heart block is well established, there is no evidence to suggest that increased vagal tone is the mechanism in the patient suffering out-of-hospital cardiopulmonary arrest (CPA). Many of these patients have initial ventricular fibrillation, which deteriorates to asystole; thus a trial of defibrillation seems indicated. What appears to be asystole in one lead may be revealed as fine ventricular fibrillation in another lead. A recent study by Coon et al<sup>24</sup> failed to show any beneficial effect of atropine in patients with prehospital cardiac arrest with asystole or pulseless idioventricular rhythm at less than 40 beats per minute. Prognosis in this group of patients remains extremely poor with mortality approaching 100%.

## Endotracheal Drug Administration

It has recently been shown that various cardiac medications are safely and effectively administered via the endotracheal route. When given as 10 ml of 1:10,000 solution, epinephrine is rapidly absorbed across the bronchi.<sup>25</sup> Other drugs which are well absorbed via this route include atropine, lidocaine, naloxone (for narcotic overdose where venous access may be difficult) and diazepam.<sup>26</sup> Drugs administered endotracheally have an extended duration of action from two to five times greater than the IV route<sup>27</sup>; this should be kept in mind when using the endotracheal route.

## Treatment of Ventricular Fibrillation

It is well established that the primary factor affecting survival in patients suffering CPA from ventricular fibrillation is the amount of time elapsed between onset and defibrillation; the earlier the defibrillation attempt the higher the chance of success. Since 60% of CPA is due to ventricular fibrillation<sup>17</sup>, the training of prehospital providers in

the technique of defibrillation is essential. Data has been presented to show the positive impact on survival of training prehospital personnel in defibrillation. An excellent study by Weaver et al<sup>28</sup> demonstrated the impact of training prehospital personnel in defibrillatory technique. With prehospital defibrillation, the rate of successful resuscitation rose from 15% to 43% in all cases of cardiac arrest and from 23% to 53% in cases where the initial rhythm was ventricular fibrillation. The American Heart Association emphasizes that electrical defibrillation should take precedence over all other modalities used in treating ventricular fibrillation, including airway management and venous access procedures.

The optimal energy levels for defibrillation have not been established. There is concern over possible myocardial damage if large amounts of current are used, although it is debatable how significant this may be clinically. A recent study by Weaver et al<sup>29</sup> showed no difference in outcome when patients were defibrillated with either 175 or 320 Joules initial energy. This is in accordance with the AHA recommendation of 200-300 Joules. Since transthoracic resistance decreases with repeated counter shocks<sup>30</sup>, the AHA recommendation of paired countershocks seems logical.

## CONCLUSION

Successful resuscitation depends upon prompt CPR to provide cerebral blood flow followed by timely restoration of adequate coronary flow. In cases of prolonged arrest, open-chest cardiac massage is probably indicated to maintain coronary perfusion. Calcium chloride has never been proven effective in the treatment of cardiac arrest; recent evidence suggests that it may be deleterious. Following current American Heart Association guidelines may produce toxic levels of serum calcium. Slow channel calcium blockers are probably useful in maintaining cerebral perfusion during CPR and may also have beneficial cardiac effects. Recommendations regarding drug therapy are controversial at this time, and are likely to change dramatically in the future.

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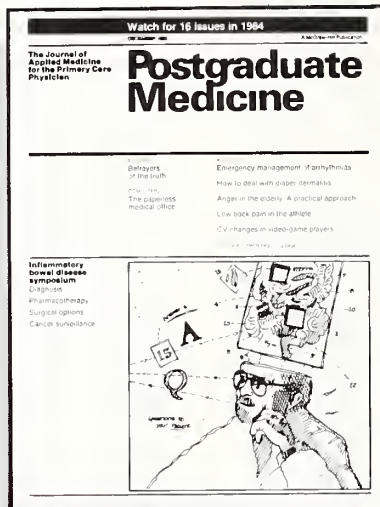
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# Cancer Trends: Hyperalimentation of the Cancer Patient

An interview with Matthew J. Lambert III, MD,  
conducted by Gerald Goldstein, MD,  
in Charlottesville, Virginia

*One of the recent significant advances in medical care has been the development of effective techniques for delivering a balanced diet by parenteral routes. Those involved with oncology and nutrition are trying to learn how to use these techniques in cancer patients.*

**DR. GOLDSTEIN:** How do you determine that a patient is in need of hyperalimentation?

**DR. LAMBERT:** Many patients with carcinoma have some element of nutritional deficiency by the time they present for treatment. Such nutritional problems may be caused by the tumor's location, such as esophageal or gastric tumors, or by the non-specific effects of neoplasms, such as anorexia. A history of recent weight loss, decreased food intake, nausea or vomiting should certainly raise

From the Department of Surgery, Division of Nutritional Support (Dr. Lambert) and the Department of Medicine, Division of Hematology-Oncology (Dr. Goldstein), University of Virginia. Address correspondence to Dr. Goldstein at Box J27, Blue Ridge Hospital, University of Virginia, Charlottesville VA 22901.

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questions regarding the patient's nutritional status. Anthropometric measurements using the upper extremity (triceps skinfold thickness, mid-arm muscle circumference) can be compared with standard values to assess the patient's fat stores and peripheral protein mass.<sup>1</sup> Routine laboratory studies, such as complete blood count, albumin, total protein and transferrin, should be obtained to assess visceral protein status. Skin testing of a battery of standard antigens assess cell-mediated immunity, which can be compromised by nutritional defects.

An appropriate level of calories and protein should then be provided to the patient and caloric intake monitored. If the patient fails to take in enough of a standard meal, supplemental protein and calories can be provided. If this fails and the patient's gastrointestinal tract is functional, enteral feedings by tube can be employed. If enteral feeding cannot be satisfactorily accomplished, then one should consider parenteral feeding to achieve the desired goal of nutritional repletion.

**What benefits are provided by hyperalimentation in the way of preparing a patient for major cancer surgery?**

It is quite clear that malnourished patients undergoing major operative procedures have a greater morbidity and mortality than those whose nutritional status is normal.

Mullen and his colleagues at the University of Pennsylvania have developed what they term the "Prognostic Nutritional Index (PNI)." This was developed using the four variables of albumin (ALB), triceps skinfold thickness in millimeters (TSF), transferrin (TFN), and delayed hypersensitivity response (DH) to any recall antigen where 0 = non-reactive; 1 = <5mm induration; 2 = ≥5mm induration. The formula is:

$$\text{PNI}\% = 158 - 16.6 (\text{ALB}) - 0.78 (\text{TSF}) - 0.20 (\text{TFN}) - 5.8 (\text{DH})$$

Patients are classified as high risk (PNI 50%), intermediate risk (PNI 40%-49%), or low risk (PNI < 40%). In a series of patients undergoing gastrointestinal operations, high risk patients had a 26% risk of major sepsis and 33% mortality, compared with 3% major sepsis and 3% mortality in the low risk group. Intermediate risk patients had a 9% incidence of major sepsis and 4.3% mortality. With regard to all complications, high risk patients had an incidence of 46%, intermediate risk 30%, and low risk 8%.

It is clear that nutritional status has a marked influence on the complications of major surgery.

#### **What do you give the patient in the way of calories, carbohydrate, protein, fat, minerals and vitamins?**

The basal energy expenditure (BEE) in kilocalories for an individual can be obtained from the equations of Harris and Benedict. These employ the age in years (A), height in centimeters (H), weight in kilograms (W), and sex of the patient and are as follows:

$$\begin{aligned}\text{BEE in men} &= 66.47 + 13.75 W + 5.0 H - 6.76 A \\ \text{BEE in women} &= 655.10 + 9.56 W + 1.85 H - 4.68 A\end{aligned}$$

Additional energy cost can be estimated based on the patient's clinical situation. For instance, patients who are resting and occasionally ambulating in the hospital have an energy requirement 5-10% above the BEE.

The protein requirement of an oral diet is estimated to be approximately 0.8 grams per kg per day. For an individual receiving total parenteral nutrition, the amino acid requirement would be 1.0-1.5 g/kg per day.

We are now recommending that patients receive approximately 30% of their daily non-protein calories as fat. Vitamins are provided on a daily basis using a standard formulation, such as MVI-12, and providing vitamin K to the patient as necessary. The importance of trace elements in parenteral nutrition has become increasingly appreciated, al-

though exact requirements are difficult to define. Standard formulation of trace elements are available in most pharmacies for intravenous infusion.<sup>2</sup>

#### **Does the cancer patient present any special problem with regard to monitoring this therapy?**

One of the major errors made in attempting to rehabilitate any patient's nutritional status is to try to do it rapidly. Weight loss and protein catabolism can be reversed with refeeding, but anabolism takes time. A cachectic individual with an energy requirement of 2000 kcal/day will not synthesize protein any faster if two or three times the recommended caloric level is given. In fact, rapid refeeding may be associated with complications, such as fatty infiltration of the liver, glucose intolerance, or profound hypophosphatemia.

Monitoring the cancer patient receiving parenteral nutrition should include electrolytes, BUN, creatinine, glucose, albumin, total protein and transferrin weekly and certain tests more often, depending on the clinical circumstances. Twenty-four-hour urine determinations for either total nitrogen or urine urea nitrogen can be obtained two or three times weekly to assess nitrogen balance. Nitrogen intake is easily obtained due to the defined amount of nitrogen in each liter of solution. Nitrogen output is subtracted and a positive (anabolic) or negative (catabolic) value is obtained. If urine urea nitrogen is measured, another 4 grams should be added to the figure to account for other nitrogen losses.

**To consider the cancer patient who has advanced cancer and is malnourished or will receive chemotherapy or radiotherapy: If we build up a patient's body mass by hyperalimentation, can the patient feel better, be more active after the hyperalimentation for days, or weeks, or longer?**

This question is difficult to answer because of the tremendous individual variability in response to a given cancer, chemotherapy, radiation therapy or nutritional support. It is reasonable to assume, however, that providing an individual with an adequate enteral or parenteral intake will allow more normal daily function than if they remained malnourished, weak and debilitated.

**Is there a home hyperalimentation technique that will enable a patient to function better?**

In-hospital nutritional support is expensive and time-consuming. For certain patients, outpatient



nutritional support is a more satisfactory method but is most easily accomplished using the enteral route. This is most commonly employed in individuals with carcinoma of the head and neck who cannot eat on their own but whose gastrointestinal tract is functional. Total parenteral nutrition (TPN) as an outpatient requires a team approach. It is probably only indicated for those patients who have a reasonable life expectancy and whose gastrointestinal tract cannot be used effectively for enteral feeding.

**Does receiving hyperalimentation make it possible to give more radiation therapy or chemotherapy?**

It has been stated that cancer patients who are well-nourished or have received TPN tolerate chemotherapy or radiation therapy better than malnourished patients. This may indeed be true, but most of the studies making this assertion are based on historical controls or controls not receiving support for various reasons.

A few well-controlled studies evaluating nutritional support and anti-cancer therapy have failed to document improvement in response to therapy or survival. But these studies likewise have numerous flaws. At present the question still has not been adequately answered, and further, well-controlled clinical studies are required.

**Does hyperalimentation improve the results of treatment of advanced cancer with chemotherapy?**

It is difficult to find studies in the literature which would support this viewpoint. As a matter of fact, as noted above, there are a few studies which have shown no significant benefit from nutritional support.

A 1979 study from Indiana University did study the effect of a 28-day course of TPN on children with advanced malignant disease. TPN for 28 days supported weight gain, increased serum albumin and transferrin to normal concentrations and reversed anergy in 64% of patients retested. However, the small and heterogenous nature of this patient population prevented reaching conclusions regarding the effects of nutrition on tumor response or survival. Further studies will be necessary to support such a thesis.

**Does hyperalimentation enable a cancer to grow faster?**

Many studies document increased rates of tumor

growth in animals receiving exogenous nutrients when compared with deprived animals. However, the use in animals of rapidly growing tumors which achieve large size in a relatively short time is not analogous to the human with cancer.

There has been no study documenting accelerated tumor growth in a human receiving nutritional support. In a study by Mullen, rates of protein synthesis in human upper-gastrointestinal malignancies were not increased by providing adequate calories and protein. This and other studies suggest that in the human, tumor growth is relatively insensitive to nutritional support. Thus, parenteral nutrition may benefit the host rather than the tumor.

**Hyperalimentation has been used at the same time intensive chemotherapy is given. The chemotherapy used interferes with DNA, RNA and protein synthesis. Is there any danger that some amino acids no longer able to be assembled into protein would be toxic?**

I am not aware of any studies documenting a toxic effect of TPN during chemotherapy. Amino acids not utilized would be degraded and excreted.

**Would you summarize your views on the indications for hyperalimentation in cancer patients?**

The use of nutritional support should be considered in any patient who cannot eat, will not eat, or cannot eat enough. It can be used to prevent nutritional deficiency or to restore a depleted individual.

Ideally, a diet can be provided which will give the patient adequate calories and protein allowing nutrition to be maintained volitionally. Supplemental feedings may be required. If the patient cannot maintain an adequate oral intake and gastrointestinal tract is functional, tube feedings should be considered. Only after these methods fail or cannot be applied should parenteral feedings be attempted. It should be reserved for those patients with treatable disease and cannot be justified as support for the terminally ill.

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# Chronic Lung Disease: Inception in Childhood

Edwin L. Kendig, Jr., MD,  
*Richmond, Virginia*

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The etiology of chronic lung disease is not always clear, but it may originate in childhood. Certain inciting agents have been recognized, and early lung insults, including smoking, may be associated with the disease. While some chronic lung disease cannot be prevented, early diagnosis will ensure proper therapy.

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From the Department of Pediatrics, Medical College of Virginia/Virginia Commonwealth University and St. Mary's Hospital, 5801 Bremond Road, Richmond VA 23221.

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CHRONIC lung disease may have, and probably often does have, its inception in childhood.<sup>1</sup> The etiology of chronic lung disease is not always clear, but it is recognized that the inciting agent may be a chemical, a hypersensitivity reaction or a viral agent.<sup>2</sup> Certain early lung insults such as premature birth, hyaline membrane disease, bronchopulmonary dysplasia, croup, bronchiolitis and adenoviral pneumonia may be associated with later manifestations of chronic lung disease.<sup>3-8</sup> One study (Tausig<sup>9</sup>) suggests that the aspiration of hydrocarbons does not result in permanent lung damage.

The early manifestations of chronic lung disease, whether in infancy, childhood or adult life, may be essentially the same: exertional dyspnea, tachypnea and cough (or cough and tachypnea).

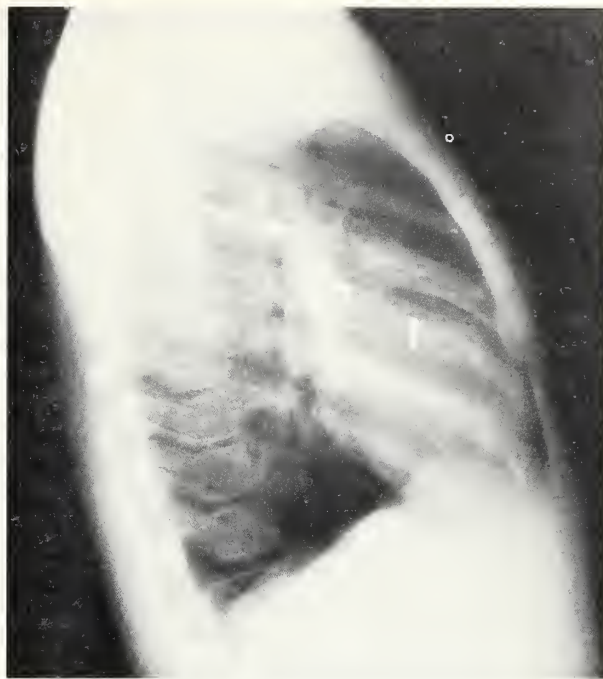
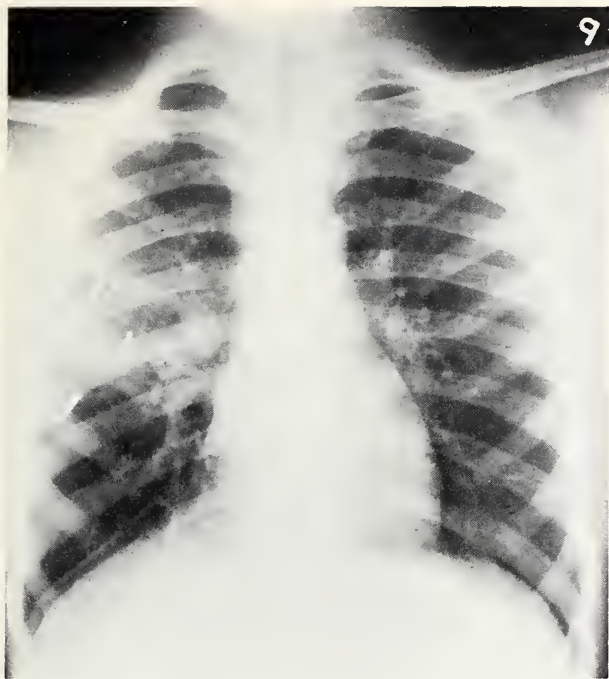
Acute bronchiolitis, a disease that always occurs in infants under 2 years of age and usually in those less than 6 months of age, presents two major problems: 1) hypoxemia, which is always present, and 2) dehydration, which may be present. The disease is characterized by an acute or even critical illness, usually no more than two to three days in duration. The dehydration, if present, is usually remedied without problem. However, hypoxemia may persist for several weeks, months or even longer, and blood gases taken from the infant at the time of discharge from the hospital will corroborate that fact. It is not always easy for the clinician to determine when to become concerned about persistent ventilatory abnormalities, but the possibility of subsequent bronchiolitis obliterans and the much greater possibility of reactive airway disease must be kept clearly in mind.

The diagnosis of bronchiolitis obliterans must be considered when 1) manifestations of the disease persist; 2) the process appears to be worsening; 3) the disease is a severe one; or 4) there is persistent evidence of a diffuse process on chest roentgenogram and/or on physical examination.

Treatment of this disease is suppressive and entails the use of corticosteroids over a varying period of time, usually for a minimum of six months.

The following patient with bronchiolitis obliterans, followed over a 9-year period, clearly illustrates the response to therapy. Diagnosis was confirmed by lung biopsy at 7 months of age and the patient was treated with prednisone, one milligram per kilogram of body weight per day for a period of six months. He appeared to be making satisfactory progress, although he had developed mild asthma. Prednisone was discontinued, whereupon the patient went steadily downhill. Six months after the





Figs. 1A and 1B. Chest roentgenogram at 9 years of age.

discontinuation of prednisone, the respiratory rate was 96/minute and blood gases showed the  $PO_2$  to be 48. Prednisone, one milligram per kilogram of body weight per day, was reintroduced, and 18 months later, after a total of two years of medication, the patient appeared essentially well. Respirations were 24 to 28/minute and  $PO_2$  had reached 90. The patient has now been without prednisone therapy for a period of six years, and except for mild asthma, appeared to be doing well at the time of his last visit in August 1983 (Figs. 1A and 1B).

Finally, a word about smoking, for which the relationship to later COPD and lung cancer has been clearly established. A survey by the National Institute of Education in 1979<sup>7</sup> indicates that 11.7% of children in the United States between the ages of 12 and 18 years (3.3 million people) are regular smokers, usually indicating daily cigarette use. In contrast to respiratory infections, smoking is a problem for which tools for prevention do exist.

In summary, chronic lung disease may have its inception in childhood. Chemical agents, hypersensitivity reaction, or viral agents may be implicated. The usual clinical manifestations are exertional dyspnea, cough and tachypnea (or tachypnea and cough). While much chronic lung disease cannot be prevented, diagnosis should be established early so that proper therapy can be instituted. An all-out effort to discourage smoking should be established.

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# Retrosternal Nipple Shadow

Morton L. Moss, MD,  
and Robert A. Ferris, MD,  
*Fairfax, Virginia*

**N**IPPLE shadows can easily be confused with intraparenchymal lesions. This is particularly true when a symmetrical nodule is not seen on the opposite side. The characteristic appearance of nipples visualized on the PA radiograph has been described.<sup>1</sup> By using similar principles defining the appearance of the nipple, the radiologist can avoid unnecessary diagnostic procedures when encountering a nipple which simulates a nodule on the lateral radiograph.

We illustrate with a case report in which a nipple masquerades as a retrosternal nodule.

## Case Report

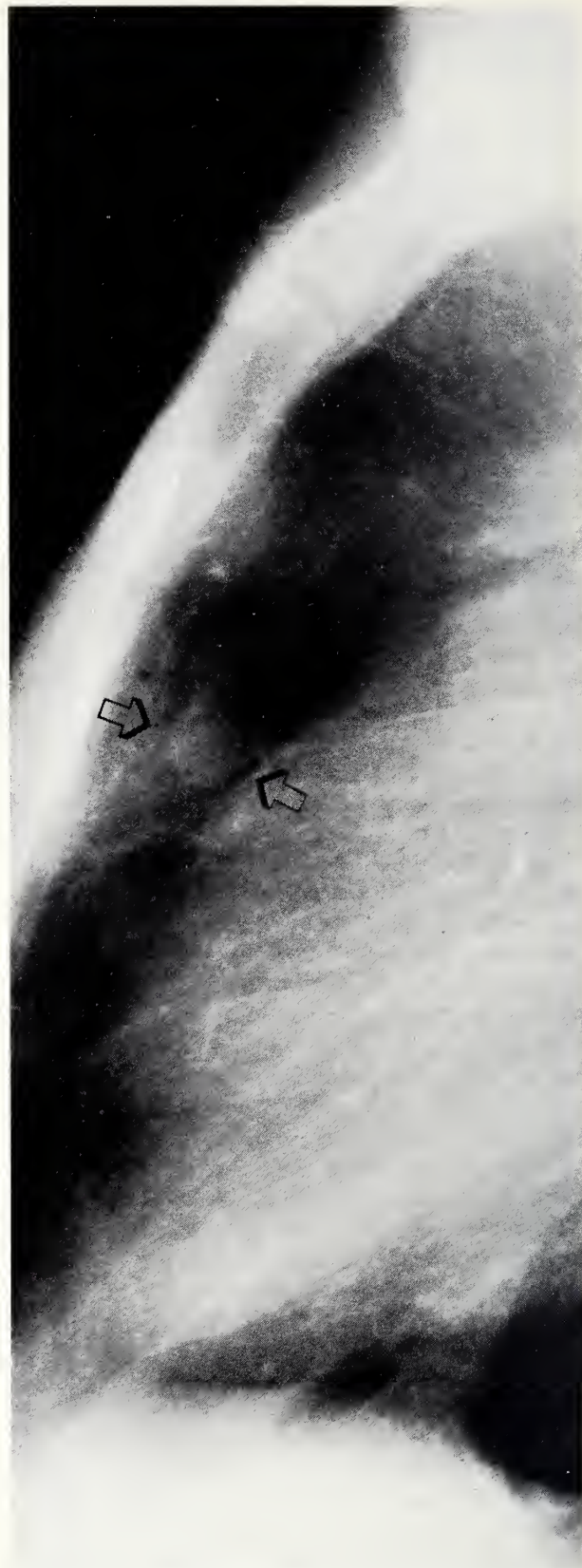
The patient, a 31-year-old female admitted to Commonwealth Hospital for acute depression, was noted to have a retrosternal nodular density on the lateral chest X-ray (Fig. 1). The PA radiograph demonstrated two symmetrical nipple shadows with characteristic sharp lateral borders and poorly defined medial margins, inferior in location to the nodular density on the lateral film (Fig. 2). Careful scrutiny of the retrosternal nodular density showed a well-defined anterior margin and a poorly defined posterior border. Its true identity was confirmed with both fluoroscopy and the use of nipple markers on the lateral radiograph.

## Discussion

Nipples visualized on the PA film commonly exhibit sharp, well-defined lateral margins and vague medial borders. This has been postulated to occur because when positioned against the cassette, the nipple usually projects laterally, creating an air-soft tissue interface. Small nipple markers have been employed on repeat radiographs to confirm the true identity of the suspected density. However, a degree of uncertainty remains, since cases have

From the Department of Radiology, Commonwealth Hospital, 4315 Chain Bridge Road, Fairfax VA 22030.  
Address correspondence to Dr. Moss.

Submitted 8-11-83.



**Fig. 1.** Nodular density projects in mid-retrosternal area on lateral view (arrows).



been documented wherein the nipple marker coincided with an intrapulmonary lesion.<sup>2-4</sup> Oblique views with nipple markers,<sup>3</sup> as well as a single view with small nipple markers and the arms extended high over the head (commonly called the "surrender view"), have been advocated. The mechanism of the surrender view, elevation and thinning of the breast tissue accounts for the current appearance of a nipple simulating a retrosternal nodule.

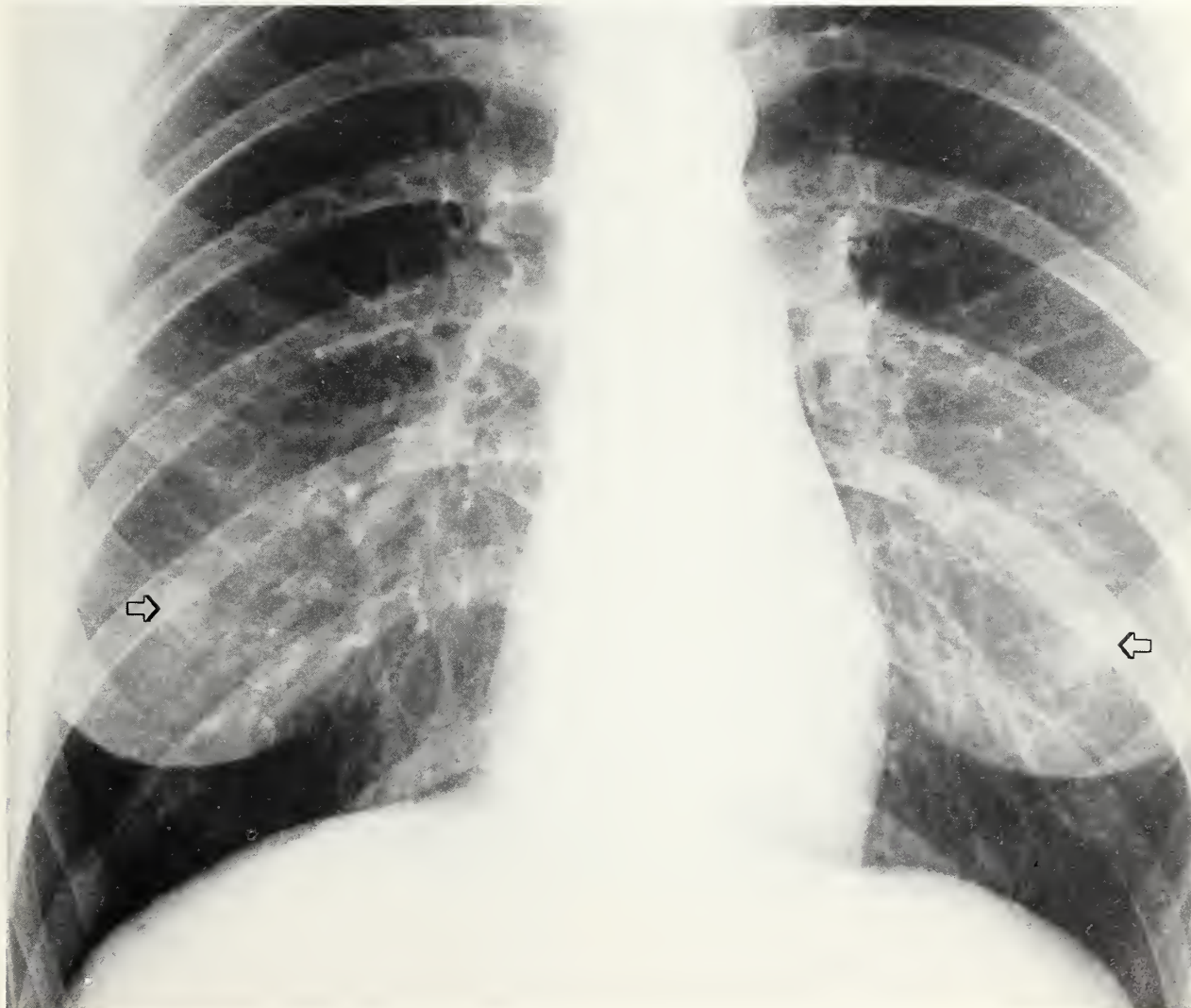
In the lateral projection, patients extend their arms above their heads, causing the elevation of the breast and muscular tissue along the anterior chest wall, with associated thinning. In this patient's radiograph, the nipple has migrated cephalad to lie at the level of the mid-sternum, with slight rotation then projecting it posterior to the sternum. When

the nipple is pressed against the cassette, it shows the same characteristics as it would on a PA radiograph, but with a sharp border anteriorly. This appearance should suggest the nipple's true identity and may be documented by chest fluoroscopy and/or a lateral view with small nipple markers. In order to remove any uncertainty of nipple markers projecting onto a true intrapulmonary retrosternal lesion, it may be worthwhile to repeat the lateral radiograph using nipple markers but with the arms hyperextended in the dependent position.

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**Fig. 2.** PA radiograph shows bilateral nipple shadows with sharp lateral margins and vague medial borders (arrows).



# VIRGINIA MEDICAL

## Harry Warthen, MD

**E**LSEWHERE in this issue is a memoir of Harry Warthen. Anyone reading it will recognize at once that his death marks the passing of a giant of Virginia medicine. Along with his many other accomplishments, he was for 20 years the Editor of this journal.

I knew Harry Warthen in a different way. When I was appointed in the late 1950s as a member of the

now-defunct Richmond City Board of Health, he was chairman of that board. From the day of my appointment to the time of his passing, Harry Warthen was my friend and benefactor. He was a warm, caring person who went out of his way to offer guidance and help to those younger and less experienced. Like many others, I shall miss him.

EDWIN L. KENDIG JR., MD

## Baby Doe: Government vs Medicine

**T**HE federal government is at it again, with yet another intrusion into medical care in the United States. If you have recently had occasion to visit a newborn nursery in any of the hospitals in Virginia, you have probably seen posted a 5" x 7" card (buff or light blue) entitled "Principles of Treatment of Disabled Infants" and proclaiming that "It is the policy of this hospital, consistent with Federal law, that nourishment and medically beneficial treatment (as determined with respect for reasonable medical judgements) should not be withheld from handicapped infants solely on the basis of their present or anticipated mental or physical impairments."<sup>1</sup> The card further states that "This Federal law, Section 504 of the Rehabilitation Act of 1973, prohibits discrimination on the basis of handicap in programs or activities receiving Federal financial assistance."

The buff card then continues, "For further information, or to report suspected noncompliance, call: Virginia State Child Abuse and Neglect Hot Line 1-800-552-7096 or US Department of Health and

Human Sciences (HHS) 800-368-1019 (Toll-free; available 24 hours a day; TDD capability)." The blue card provides space also for the insertion of the telephone number of an Infant Care Review Committee (recommended as an addition by the American Academy of Pediatrics). Both cards end with the note that the "identity of callers will be held confidential" and warn that federal regulations prohibit retaliation by the hospital.

This is the second set of directives from the Department of Health and Human Services on this subject and has been modified from the first as the result of legal actions by the American Academy of Pediatrics, along with the American Medical Association and other organizations. (The AMA has now filed suit against HHS Secretary Margaret Heckler, seeking to have the final version of the regulations declared invalid.) While the first rule was in effect, examples of the seriousness of intrusion into patient confidentiality and medical care were well documented.<sup>2</sup> The second version of the HHS rule, now in effect, is not significantly different from the first,





HARRY J. WARTHEN JR., MD  
EDITOR, 1956-1976, VIRGINIA MEDICAL MONTHLY

in that it is based solely on the concept that federal law enforcement activities are necessary to ensure against inappropriate treatment of seriously ill newborns. It does, however, encourage (but does not require) hospitals to establish infant care review committees (ICRC) and to establish policies on medical treatment of handicapped infants.<sup>3</sup>

This major controversy as to responsibility for patient care was generated by the "Baby Doe" case. In Bloomington, Indiana, in the spring of 1982, a term male infant with Down syndrome, esophageal atresia, and a tracheoesophageal fistula was born. The parents decided to forego treatment and the infant was allowed to die.<sup>4</sup> In the process, an intensive medical, legal, and ethical debate ensued. The hospital administrators sought legal advice and several lower court hearings and a hearing by the Indiana Superior Court were held.<sup>5</sup>

Much has been written<sup>6-9</sup> on this subject, and the answers are not easy. As the president of the American Academy of Pediatrics has pointed out,<sup>10</sup> the pediatrician's primary responsibility is to the child, and decisions in the newborn nursery should be made on the basis of what is best for the child. While the views of the family, especially the parents, must be considered, economic, psychologic, or other family considerations should not determine the course of treatment.

Perhaps the fundamental issue is best presented by Angell,<sup>11</sup> who writes that it "is whether judgments about the treatment of handicapped newborns ought to be made by the State. The usual intent of rules against discrimination is to protect individuals from malice or indifference. In the case of handicapped newborns, it is difficult to imagine people who are *less* malicious or indifferent than parents and physicians. Probably most parents would give their lives for their children; the circumstances in which parents would prefer death to survival of their child must be extraordinary indeed. For their part, physicians are accused of many wrongs in our society, but lack of therapeutic aggressiveness is seldom one of them. Indeed, physicians as a group are considered to be almost mindlessly devoted to keeping life going at all costs. Yet the Baby Doe rules seem to depict parents and physicians as adversaries of the infant who should be removed from decisions about whether to treat life-threatening disorders."

Now Congress is in on the act. Baby Doe provisions have been introduced as part of both House and Senate child abuse legislation and HR1904 has been passed by the House. As this is written Senate Bill 1003 is still under consideration. It is significant

that HHS is opposed to Baby Doe provisions in legislation, believing that the present regulation is more than adequate. From the physician's viewpoint, no government regulation seems necessary.

EDWIN L. KENDIG, JR., MD

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## Challenging Unsettled Times

**I**t is a real challenge to lead the Richmond Academy of Medicine when changes are taking place so rapidly in the health care system. Changes can be both frightening and exciting. There were exciting changes in medicine in 1983. These were matched by some unsettling changes in several reimbursement systems. How we in 1984 and in years to come react to these changes will shape our role in the entire health care system. We should discard the

Presented as the inaugural address of the president before the Richmond Academy of Medicine on January 10 at the Academy's headquarters in Richmond.



notion that if we fight tooth and nail to maintain the status quo, we will succeed in retaining our current role. We must prepare for changes and adjust so that we can provide good care to our patients and still be adequately compensated.

I am often asked whether or not there is still a need for the Academy. The Academy began as physicians united against threats to the proper practice of medicine. Now physicians are feeling threatened by the establishment of new payment methods, such as PPOs, HMOs, DRGs, and are separating into groups for protection. Competition is intense and we are competing against each other for the same dollar. More division will occur as each hospital and insurance company establishes its own PPO. Soon we will have many islands of physicians competing financially and medically. It is very important that a strong and active Academy continue to exist. We will represent all physicians no matter with whom they are allied. The Academy will continue to be the hub that is needed to keep us together. It will also continue to remind us that our primary goal is the practice of medicine and that our patient's well-being is the Number One priority.

I see the Academy for the next few years becoming more political. To facilitate this I hope to expand the education of our members, to increase public relations and to have a more forceful and visible legislative committee.

I feel most fortunate to succeed a president who has seen the necessity of changing the Academy's role to a more active one. The Law and Economics Health Care Commission which he established has produced an excellent report which we will be using. I would like to commend Dr. Davis on the past year's accomplishments. I will continue the programs that he has initiated.

I plan for the Academy to continue to educate us about the new health care alternatives. New HMOs announced their intention to seek providers. Still more groups will appear this year. I hope to provide you with the knowledge that you will need to make a decision concerning these new alternatives. The Program Committee for 1984 has been asked to seek topics to prepare us for the changes to come.

We plan to monitor federal and state regulations so that we can anticipate and alert our members to threats against our profession. If we present a united effort, our patients will be the beneficiaries. A joint committee with the Dental Society has been established and it will be chaired by Dr. Norman Sporn. It will investigate and study issues of mutual concern and report the findings to you.

The Cost Containment Committee has been di-

rected to continue to gather information about our practice trends and to distribute this to you.

Information will be dispensed to the members by a newsletter which will be issued at least monthly and more frequently if needed. I hope to keep our members abreast of major happenings not only in the community but in the state and nation. As we have seen many times in the past, what happens in California and Massachusetts will eventually need to be addressed in Virginia. If we are aware of all the issues, then we can shop wisely both for ourselves and for our patients. The monthly bulletin will continue in the same format and will list membership changes, meetings, Board reports and other general Academy business. I want you to stay close to the Academy and I want you to know whatever we know.

Public relations is one of the most important roles. It is imperative that our message be delivered to the public. We should not rely on other people to do for us what is our responsibility. I firmly believe that the media is willing to assist us in delivering our message. They need, however, to know where to turn for the information they require. We have already started talks with various members of the press, radio and television, and hope that they will feel free to call on us and we on them.

Our participation either individually or together in many community projects is important to their success. The community, however, is largely unaware of the participation of physicians. This year we surpassed our United Way goal. Many of our members donate their time to worthwhile programs. The physician community should be recognized for this and I am asking you to supply us with the information about yourselves so that we may use it in our public relations program.

I plan to stress the advancements that have been made in the field of medicine. In the past we have been reluctant to talk about our achievements. We must get this information to the public now. These achievements have prolonged life and made it more useful, but with them comes a huge increase in the cost of medicine and the public should know this.

It is also quite important that the consumer realize that we in medicine are trying to keep costs down. We have been told in the past that if we did certain things health care costs would fall. We have initiated and accepted such cost-saving ideas as one-day surgical programs, pre-admission testing, shorter length of hospital stays, and more outpatient work, yet the costs continue to rise. The public should be informed of our efforts and realize that we alone do not hold the key to cost savings. Physi-

cians have participated in the certificate of need program and again the public should know. Our consumers should be told frequently that we are interested not only in their health but in less costly ways to provide it. For too long we have been accused of being the sole reason for the continuing rise in medical costs. We must remind our population that patient demand for convenience, greater amenities, and more extensive testing plays a great role in increasing costs. Additionally, we should make wider distribution of the fact that the physicians' share of the cost of medicine has actually decreased annually since 1965, and although physicians fees have increased, the percentage of the total cost of medicine received by MDs is less.

We should inform the consumers of the new methods of medical care delivery. You know how difficult it is for you to understand an HMO or a PPO. Imagine a patient trying to understand it. The consumer should be aware of the advantages and disadvantages of a particular system. He should know beforehand that an HMO may limit his choice of physicians or that another system may limit his choice of hospitals. He should realize that these systems are all designed to save money and that he will have certain obligations to fulfill and that his own choices will be decreased.

The consumer should also be made aware of the changes in Medicare and Medicaid reimbursement so that he will not be surprised by his decreased coverage. The consumer must realize that the changes originate from the insurers or from the government. The physician should not be placed in the position of apologizing for the patient's insurance coverage or lack of it. We should make certain that our patients understand the benefits they are purchasing.

The health care cost now has risen to 10% of the gross national product. Many people say that it is too much. I feel that it is not enough and certainly we do not need to apologize for it. For a very modest rise we may achieve such breakthroughs as finding the cause of cancer, discovering better treatments for stroke, or curing arthritis. Compare this 10% of gross national product with 17% of gross national product spent for sports and recreation. I ask you, which is the better buy for our country?

You have already voted a dues increase primarily to finance a public relations program and I assure you that it will be used efficiently. We are making every effort to deliver our story and will be asking for your help in providing us with ideas and information that you think are newsworthy. Your Academy is "going public." Dr. Kinloch Nelson has

agreed to chair the Public Relations Committee.

Probably our most important goal should be our efforts to influence legislation that we consider important. I hope to make this Academy an organization that is respected and solicited for advice by the General Assembly. This will be an ongoing commitment and cannot be accomplished in one year. Too often proposed bills go unchallenged or incorrect information is presented when we are not in attendance. In the past we thought it not in keeping with our profession to be visible in the legislative process. We can give expert testimony and our presence at the General Assembly is a major goal that I intend to pursue vigorously. Our message must be delivered and no one can do it more effectively than we ourselves. The best spokesperson for a physician is a physician.

Physicians must be represented at the General Assembly daily. I am asking each member of this Academy to give to me and to medicine his most valuable asset—his time. I would like for each member to donate one day a year for legislative promotion. I plan to have us represented daily at the General Assembly so that no issue can go forth without our knowledge and participation. We are not experts in law but neither are the legislators experts in medicine. We need each other's help.

There are several other Academy issues that are important. The desire of many physicians to advertise continues to raise many ethical questions. Advertising will increase and is considered ethical if it is not misleading. Please be careful about what you say to the public.

Additionally, I hope to increase our membership. I have met with Dr. Marcella Fierro, our Membership Chairman, and we are attempting to identify those physicians who are not members and solicit them.

In closing, I would like to say that I want to make this a more forceful, open and active Academy. I hope to involve more of our members in its activities. I hope that we will be more articulate both scientifically and politically. The next several years will be difficult. We must continue to find strength in our numbers and beliefs. I again want to remind you that we are physicians, and ultimately all decisions must be made with our patients in mind. At no time should we jeopardize our patients' care. Our patients' well-being must remain our primary goal.

WYATT S. BEAZLEY III, MD

425 North Boulevard  
Richmond VA 23230



# VIRGINIA MEDICAL OBITUARY

## Memoir of H. J. Warthen, Jr. 1901-1984

*By John P. Lynch, MD, William H. Harris, MD,  
and E. Randolph Trice, MD*

Our distinguished colleague, Harry Justice Warthen, Jr., MD, died after a long, distressing illness on March 5, 1984. Our loss is great because of his manifold talents as surgeon, historian, editor and, for 25 years, the Richmond Academy of Medicine's curator. There have been few who have matched his service to his patients, his profession, his community and his church.

He was born September 30, 1901, in Port Norfolk, Virginia, and lived part of his early childhood in Alexandria, Virginia, next door to the home where Robert E. Lee once lived. Perhaps his interest in history and that of the Civil War in particular was inspired by this circumstance.

In 1925 he received his degree in medicine at the University of Virginia, interned at St. Elizabeth's in Richmond and became resident surgeon at Johns Hopkins Hospital 1927 to 1932. He then did research in Germany from 1932 to 1933 and returned to Richmond, where he established an extensive and fruitful surgical practice until his retirement in 1972, a span of almost 40 years.

During World War II he served as a lieutenant colonel in the United States Army Air Corps, becoming chief of surgery for the Army facilities in 20 states and riding a B-17 bomber, as he recalled it, to make "house calls."

He became president of the Richmond Academy of Medicine in 1949, where he served with his usual distinction.

In 1955 to 1960 he held public office as chairman of the Board of Health of the City of Richmond, where he had served as a member since 1950.

From 1956 to 1976, a span of 20 years, he served as Editor of the VIRGINIA MEDICAL MONTHLY, now VIRGINIA MEDICAL. His whimsical, dry humor was the hallmark of his amazing total of 206 editorials, of which 46% were aimed at socialized medicine. He championed the role of the bedside nurse, medical education and, of course, the University of

Virginia, where he served as president of the Medical Alumni Association.

As curator he directed medical exhibits at the Richmond Academy on the 350th anniversary of Jamestown in 1957, the Civil War exhibit in 1961 to 1965, and the Bicentennial exhibits in 1976.

His civic and historical interests included the Monument Avenue Preservation Society, Historic Richmond Foundation, Antiquarian Society, and the Richmond Architectural Review Commission.

He was one of the principal founders of Richmond Memorial Hospital and became first chief of the department of surgery and later chief of staff.

He was a member of Alpha Omega Alpha, Sigma Xi, Nu Sigma Nu, Southern Surgical Association, American Surgical Association, diplomate of the American Board of Surgery and the American Board of Plastic Surgery, The Medical Society of Virginia and the Richmond Academy of Medicine.

He was an elder in the Grace Covenant Presbyterian Church and fought for the conservation cause for the Presbyterian Church in the United States, also known as the Southern Presbyterian Church.

He is survived by his wife, Mrs. Martha Winston Alsop Warthen, and three sons, Harry J. Warthen, Benjamin Warthen and George A. Warthen.

## S. Dawson Theogaraj, MD

Dr. Sam Dawson Theogaraj, professor of plastic surgery at the Medical College of Virginia, died at the Medical College of Virginia Hospital on March 26. He was 50 years old.

A native of Kuala Lumpur, Malaysia, Dr. Theogaraj was graduated from Christian College in Madras, India, and received his medical education at Christian Medical College of Vellore, India, graduating in 1957. Following surgical training in Vellore, he came to the United States and completed his residency at the Hospital of the University of Pennsylvania in 1966. Dr. Theogaraj returned to India and was chairman of the Department of Plastic Surgery at the University of Madras until 1972, when he came to the MCV faculty.

His wife, Dr. Janaki Theogaraj, practices child psychiatry in Richmond.

# VIRGINIA MEDICAL CLASSIFIED

*Virginia Medical classified ads accepted at the discretion of the Editor. Rates to Medical Society of Virginia members: \$15 per insertion up to 50 words, 25¢ each additional word. To non-members: \$30 per insertion up to 50 words, 25¢ each additional word. Deadline: 5th day of month prior to month of publication. Send to the Advertising Manager, 4205 Dover Road, Richmond VA 23221.*

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**MEDICAL DIRECTOR** sought to function as regional medical administrator with Virginia Dept. of Health for Southwestern Virginia. Includes 9 district offices, numerous local offices, regional office in Roanoke. To plan, coordinate delivery of medical and environmental health services. Develop policy, negotiate regional budget with local offices, other financial management activities. Also program planning, evaluation, personnel management. Must be Virginia licensed or eligible, preferably with MPH,

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**EMERGENCY MEDICINE**—Full-time positions available in four emergency departments located in eastern, central and western Virginia. Competitive income and professional liability insurance provided. Reimbursement for ACLS and ATLS training, CME tuition, ACEP dues. For details respond in confidence to: Katie Sherrill, Spectrum Emergency Care, Inc., 1111 N. Westshore Blvd., Suite 211, Tampa FL 33607, (813) 870-2356.

**FOR RENT**—Hilton Head. 3 BR, 3½ bath home with unlimited use of private pool and tennis in complex. On 18th green of beautiful Shipyard golf course and across road from golf clubhouse. Ocean is minutes away via bike, car or walkway. 2 BR villa for sale in Shipyard by same owner. (804) 874-4428.

**SAILING**—Chesapeake Bay. Charter Vixen 34' sloop, "Windrush," sleeps five. Bareboat from Deltaville. Well appointed, full electronics. Experienced sailors only. Week-end or weekly rates, April through November. Dozier's Dockyard, Rt. 33, Deltaville VA 23043, (804) 776-6711. A beauty!



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# LETTERS

## Three health administrators respond to articles on nursing homes

I sent a copy of the March issue of VIRGINIA MEDICAL, with its cover story titled "Notes on Nursing Homes from Medical Directors," to the Hon. Margaret M. Heckler, Secretary of the Department of Health and Human Services, along with the following letter:

*The March issue of VIRGINIA MEDICAL is enclosed. Please take note of the comments on pages 141-142 about the frequency of required physician visits. We have written letters over the past years concerning these requirements that are wasteful of taxpayers' money and physician time.*

*In October 1981 the Virginia State Board of Health amended the [nursing home] licensure regulations by changing the requirement of private-pay patient visitation by a physician every 90 days to state that each private-pay patient shall be seen by an attending physician as deemed necessary by that attending physician. Required visits by physicians should be reduced in frequency, and, hopefully, quality evaluations would be the result, rather than "routine as required." The physician and nurse should be free to exercise their professional judgment. Please review these requirements because better care and less cost should be the result.*

I am so pleased with the March issue. I shared it with many of my friends and Board members interested in the care of the elderly. This is another outstanding issue of VIRGINIA MEDICAL covering a very important subject of social interest. Congratulations!

**James B. Kenley, MD**

Commissioner, State Department of Health  
109 Governor Street, Richmond VA 23219

**Editors' Note:** Dr. Kenley's letter to Secretary Heckler was answered by Henry R. Desmarais, MD, director of the Bureau of Eligibility, Reimbursement and Coverage, who wrote as follows:

*We are very much aware of the concerns associated with the statutory requirement regarding the*

*recertification of Medicaid patients. The Office of the Inspector General for the Department of Health and Human Services has recently addressed this issue in an audit report and . . . [the Department] recommended the repeal of Section 1903(g) of the Social Security Act, including the certification and recertification requirements. This proposal is currently under consideration by the Congress.*

Thank you for the March issue of VIRGINIA MEDICAL. I read and enjoyed most of the articles, particularly those dealing with nursing home topics. If medical schools would only devote more time to training physicians on the "how to's" of caring for the elderly!

Marian E. Foltz

Administrator,  
Susan B. Miller Nursing Homes, Inc.  
PO Box 366, Woodstock VA 22664

Thank you for your prompt attention to my request for additional copies of the very informative March 1984 VIRGINIA MEDICAL, with its articles on nursing homes.

I would like to call attention to the statement on page 145, "Intermediate care . . . for persons not capable of living by themselves . . ."<sup>1</sup> Our local Nursing Home Preadmission Screening Committees (for current or anticipated Medicaid recipients applying for admission to a nursing home) frequently have to deal with this definition of "intermediate care."

We try to reserve Medicaid payment for intermediate care for persons who need some nursing care other than just supervision and assistance in the "activities of daily living", but who, as the above statement continues, are "not sick enough to need 24-hour nursing care." There are criteria to assist in deciding whether an applicant fits this category.

If the person needs ordinary non-professional assistance, nursing home admission is not recommended, and the family or responsible agency is urged to arrange appropriate care. Such decisions are sometimes difficult for the family, and even the



physician, to accept, because intermediate care is often considered to be for "persons not capable of living by themselves."

Perhaps sometime we will have a system of long-term care in which everyone is cared for at the level most appropriate to the need. This VIRGINIA MEDICAL issue should help Virginia physicians not already involved with such patients to appreciate the needs.

**Joanna M. Owens, MD**

Director, Middle Peninsula Health District  
Saluda VA 23149

1. AG: Virginia's old folks at home. Va Med 1984; 111:144-145

### **Likes to read practical items that "make the job a little easier"**

When looking at VIRGINIA MEDICAL recently, the thought occurred to me that there might be a one-page section titled something like, "Gems and Shortcuts." This would be an area for little items from practitioners that make the job a little easier or give a simple solution to a problem.

I know that at the University of North Carolina Medical School there has been a session each year in which faculty members presented "Gems." These were three-minute presentations of small facts, physical findings, lab reports that might be of significance clinically.

"Shortcuts" could relate to office management, such as recordkeeping. For instance, I find a red-ink pen of great help. I use it to underline important events, dates, special examinations or findings in my office charts and also on hospital charts. Frequently I mark PDR or phone numbers with red ink. It is amazing how many times one goes back to the same phone number or medicine reference, and it is so much easier to find it the second time when it is marked in red.

Another shortcut is the use of a marker card that is placed in the patient's file when a chart is removed and the patient is being seen. The cards are cut from x-ray cardboard to about  $8\frac{1}{2} \times 11$ , and yellow plastic tape is placed on the edge. The card is placed in the file with the yellow tape showing; this enables one to replace the chart with ease, rather than having to spend a lot of time thumbing through the file to arrive at the proper space.

Maybe the above would not appeal to many people, but I find that I remember some of these short items, when I see them, much more readily than I do the detailed articles which we must now attempt to read in order to "keep up."

**Roger M. Winborne, Jr., MD**

374 Woods Avenue, SW  
Roanoke VA 24016

### **Opposes a senator's stance on anti-tobacco bills**

I wish to bring to the attention of the medical community of Virginia statements made by Senator Paul Tribble that appeared in his newsletter published in February of this year, entitled "U.S. Senator Paul Tribble Reports." In an article entitled "Opposes Anti-Tobacco Bills", Tribble states that "these efforts [to increase required warnings about the dangers of smoking] represent an unwarranted governmental intrusion into the private sector and into our lives."

He continues, "I will vigorously oppose these bills in the committees of the Senate and on the floor of the Senate. It is time to end the federal war on tobacco and let Americans make their own choices to smoke or not to smoke, free from the heavy hand of federal legislation."

I find these statements outrageous and shocking, as I expect most people in the medical profession do. We all understand that smoking is the major *preventable* causative factor of cancer and coronary artery disease. Cigarette smoking is a major contributor to the mounting cost of health care. It figures heavily in loss of life and property through fires and in decreased work efficiency. A vigorous public policy to decrease cigarette smoking is Senator Tribble's responsibility.

I urge all physicians to let Senator Tribble know that his blind support of the tobacco industry to the exclusion of reason and compassion is untenable. I further urge the members of the Virginia Medical Political Action Committee to withhold support from Senator Tribble and other candidates who so blatantly oppose basic public health common sense.

**Martin Albert, MD**

211 East High Street  
Charlottesville VA 22901

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## ACTIONS OF COUNCIL

Here are some of the matters that engaged The Medical Society of Virginia's Council when it met on April 14 at MSV headquarters in Richmond with President C. Barrie Cook presiding.

● **Plans.** The councilors saw architect's plans for a proposed expansion of the attic area that would add 1,750 square feet of office space to the Society's headquarters building. In effect, a second, dormered floor would be added. The proposed expansion was approved.

The Long-Range Planning Committee is considering a survey of Virginia physicians, reported chairman William J. Hagood, Jr., in quest of information to help the committee analyze the Society's current position and future goals. The councilors liked the proposal and asked Dr. Hagood to proceed with the survey.

● **Peer Review.** Formal application for designation of the Medical Society of Virginia Review Organization as the official peer review body in Virginia was about to be transmitted to the Health Care Finance Administration, the councilors were told by Freeman H. Vaughn, MSVRO consultant. Mr. Vaughn expressed expectation of an affirmative answer by early this month.

● **Membership.** Dues-exempt membership was the subject of a recommendation brought to the councilors from the Membership Committee by Chairman J. Thomas Hulvey. Lower the age for dues-exemption to 65, the committee recommended, if the member of that age is fully retired and has been an MSV member for at least ten years. The Council approved.

A second recommendation asked for addition to the Society's House of Delegates of a hospital medical

staff section, similar to the one established last year by the AMA. Again the councilors assented, and they set the section's organizational meeting for November 8 in Williamsburg, just before the House convenes for its 1984 session.

● **Tax Exemption.** Should organizations with tax-exempt status operate closed-panel alternative delivery systems that may benefit from the exemption? The Council's consensus view was negative. An ad hoc committee of these three physicians is looking into the matter: Dr. Glenn B. Updike, Jr., Dr. Frederick K. McCune, and Dr. Wyatt S. Beazley III.

● **DRG Certification.** Sharply questioned by the councilors was the Health Care Financing Administration's physician certification form for prospective payment by diagnostic-related group. The councilors discussed a letter on the subject from Dr. James H. Sammons, executive vice president of the AMA, to Carolyn K. Davis, HCFA administrator. It read in part, "Because of the questionable value and need for certification as now required and because of serious concerns about what the physician is expected to certify, we need to explore . . . whether this regulation can be withdrawn."

It was the Council's decision that The Medical Society of Virginia transmit to the AMA a communication stressing the urgent need for changing the regulations. It was further agreed that should the AMA not be successful in effecting a change, the Society should explore the possibility of legal action by way of an injunction.

For a copy of minutes detailing the meeting, write to James L. Moore, Executive Vice President, at MSV headquarters.



## From the President: What's Past Is Prologue

**A**s a pathologist and not a clinician, I am not surprised at the changes affecting physicians taking place today because clinicians are experiencing the problems many pathologists and other hospital-based specialists experienced in the past.

Pathologists were among the earliest who received all or part of their income from hospitals. Today approximately 20%-25% of physicians are in this category, including emergency room physicians, neonatologists, cardiologists, internists and others. Obviously, DRGs and other societal and governmental requirements will have an effect on their incomes and on how they will deliver the care they give.

Sixteen years ago our private laboratory billed patients directly since this was the traditional thing to do. However, in order to stay in existence and remain competitive, we had to change our billing procedures, like it or not.

No one likes change, especially physicians, and changing our billing procedures really represented a major readjustment for our laboratory and for us psychologically.

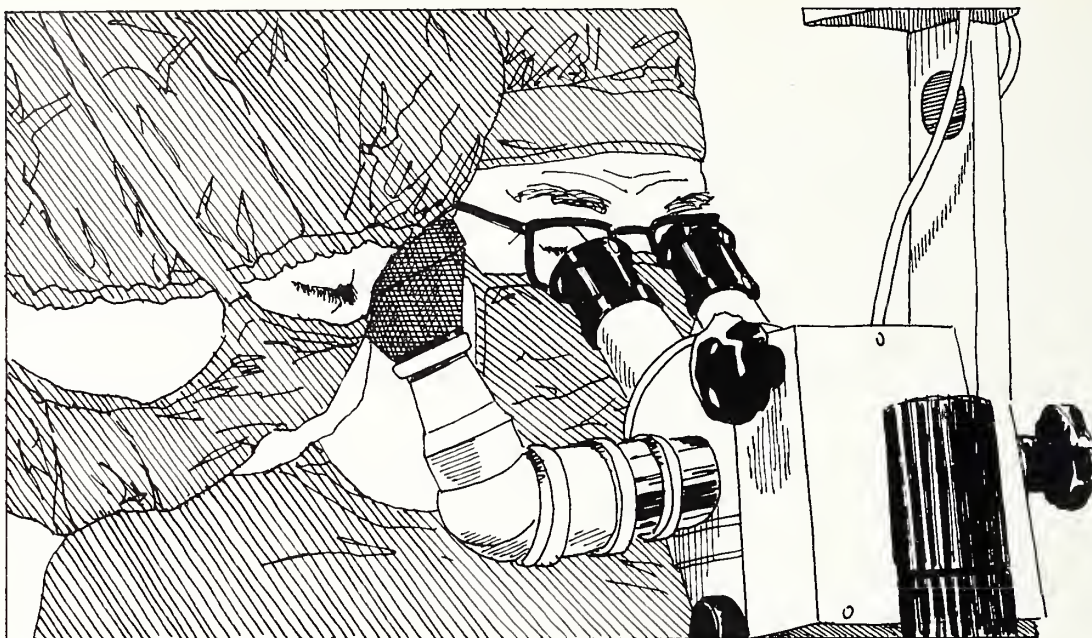
Now, 16 years later, physicians in general find themselves facing major changes in how they will practice: Will we be able to compete and, if so, how? Should we advertise or not? Do we join HMOs and just do what we like best—practice medicine? Should a group of physicians with similar types of practice form a loose amalgamation in order to have the services of a business manager, public relations firm, or what have you?

These are major decisions that you will be facing. They are not pleasant, but ones you cannot escape unless you are approaching retirement, and then you are able to remain an iconoclast to the end.

What has happened to pathology and the other hospital based specialties in the recent past soon may be happening to you. We need to be supportive of one another whenever possible in order to continue the free enterprise system of health care delivery for all physicians.

While change may be inevitable, it is the responsibility of all physicians to help assure that any evolution which occurs does so with minimal impact on the standards of excellence that have enabled our country to provide the finest quality of care in the world.

**C. Barrie Cook, MD, President,  
The Medical Society of Virginia**



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**Phillip F. Pierce, MD**, Infectious Diseases, 4731 North 23rd Street, Arlington VA 22207

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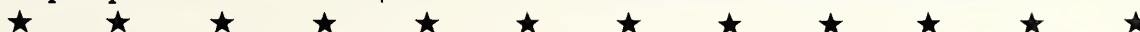
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# For the new contracts, some preventive medicine

**S**INCE the beginnings of medicine, a unique kind of contract has been the foundation of the physician's practice. Dr. Arnold Leaf calls it a "hallowed" contract and describes it like this:<sup>1</sup>

*... Only the patient, or the patient's family, and the physician are involved; no other party or parties should intrude. The contract between physician and patient is a private affair and no one else's business. The physician makes the diagnosis and, with the informed consent of the patient, manages the treatment. The patient subsequently pays the physician's fee, and that fulfills the contract.*

Now into medicine comes an entirely different kind of contract. It is all business. And it is a legal instrument, with the force of law; its terms imprison the signator within specific obligations. It is the contract that makes the physician a provider for an alternative delivery system.

Suddenly there are a lot of alternative delivery systems in Virginia,<sup>2</sup> and lots of Virginia physicians are being approached to sign their contracts. To help physicians interpret the "fine print" of such contracts, The Medical Society of Virginia's President, Dr. C. Barrie Cook, commissioned the booklet bound into this issue at page 334. It was prepared by Sandra L. Kramer, the Society's associate legal counsel, and VIRGINIA MEDICAL's editorial staff, who acknowledge with appreciation the prototype "Physician's Contracting Handbook" published by the California Medical Association. To complement the booklet, VIRGINIA MEDICAL brings you Bak and Share's guide to the advantages and disadvantages to physicians of HMOs and PPOs.

In Virginia, these new enterprises are locating largely in areas whose populations offer plenty of room for commercial growth: the capital city of Richmond and the Tidewater megalopolis of Norfolk, Virginia Beach, Portsmouth and Chesapeake. In Upper Virginia, a new HMO/IPA is challenging the five alternative delivery systems headquartered across the Potomac in Washington. In Virginia's Southwest, some Roanoke physicians are launching a PPO. (For an editorial by a Roanoke doctor, see page 257 of this issue).

For doctors in these areas, contract medicine is of immediate concern; for others it may simply be a matter of time. "It'll be several years before we get around to the community hospitals," a spokesman for an expanding HMO told VIRGINIA MEDICAL's reporter.

Already contract medicine has breached the sheltering walls of one of Virginia's three medical schools. Physicians who teach and practice at the Medical College of Virginia are now under contract as providers for KeyCare, the PPO that came on line last year under the auspices of Blue Cross Blue Shield of Virginia.

The Eastern Virginia Medical School faculty is entirely voluntary, so there's no contracting under way on that campus. Westward, at the University of Virginia Medical Center every day brings new rumors of impending HMOs and PPOs, but to date contract medicine has not materialized in Charlottesville. Will it come? When asked that question, a senior professor smiled grimly and answered: "It's like watching the lava moving slowly down Mauna Loa. You don't know whether it's going to hit you or not. All you know is that it's going to be destructive."

The perception that contract medicine threatens accustomed ways of practice is widespread in the medical community, yet many physicians fail to scrutinize the contracts they sign to see precisely what the terms demand of them.

- Item: Disenchanted with an IPA after a year's service, a physician tried to terminate the contract but found he was locked into it for three years. He had to stay.

- Item: A group of physicians fought their way out of an HMO only to discover they were liable for specialists' fees their orders had incurred. It cost them plenty.

To prevent such unpleasant surprises, the wise physician does some investigating before he signs on the dotted line. "A Guide to Physician Contracts" can help in that investigation. —A.G.

1. Leaf A. The doctor's dilemma—and society's, too. N Eng J Med 1984;310:718-721

2. AG. It's spring and the HMOs are sprouting. Va Med 1984; 111:180-183



## Contract Medicine: What's in it for you?

Brafford Bak, JD, LLM, and Scott H. Share, MBA

**M**ANY Virginia doctors, especially those who practice near major metropolitan areas, either have been or will be solicited to sign contracts with alternative health care delivery systems. Roughly speaking, two types of these systems are operating in Virginia—health maintenance organizations (HMOs) and preferred provider organizations (PPOs). If the system is an HMO, it will often be allied with an independent practice association (IPA), which would in such a case be the entity that offers the doctor a contract. PPO contracts come in a variety of forms.

You and your legal advisor should carefully review any contract before deciding whether or not to sign it. The brochure bound into this issue will help in that

Mr. Bak is an attorney and Mr. Share a consultant with the Health Care Group, Bala Cynwyd, Pennsylvania. Address correspondence to Mr. Bak at 400 GSB Building, One Belmont Avenue, Bala Cynwyd PA 19004.

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examination—but it is no substitute for specific legal guidance. This article will give you some general information about HMOs and PPOs—emphasizing the advantages and disadvantages of contracting within the two types of systems.

### HMOs/IPAs

Health maintenance organizations have been in operation in this country for 25 years; thus most physicians are conversant with their characteristics.

Generally, HMOs are distinguished by their “capitation” method of payment to primary care doctors, and sometimes also to specialists, and by their requirement that subscribers use only providers who contracted with their HMO or an allied IPA.

Contracting with an HMO or an IPA may or may not leave a doctor the option to treat patients who are not HMO subscribers. At an extreme, “group practice” HMOs or allied organizations simply hire doctors as full-time employees at a

flat salary. Other, “closed panel” HMOs look to a small number of providers, not letting other doctors participate, except as policing of the ranks eliminates over-utilizers.

The capitation payment system—most frequently a fixed amount per month per subscriber enrolled at the beginning of each month—has the advantage to the HMO organization of cost containment. The disadvantage to the doctor is that a subscriber, generally not restricted by any co-pay requirement, is not motivated to limit his or her use of health care services. The doctor is therefore at financial risk if a subscriber chooses to over-use services. The risk of this switch of the “insurance” function is very real. For example, a recent article in *Medical Economics*<sup>1</sup> by a family physician describes how the 20% of his group's patients who were HMO subscribers took up more than 50% of the group's appointment time. Other groups have reported similar experiences.

A different HMO/IPA approach

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***"It may be a way  
to compete  
more successfully."***

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to paying doctors is to pay both primary care doctors and specialists on a fee-for-service basis, but to withhold a percentage from initial payments. Some or all of that "hold-back" is later paid to the doctors, but only if their utilization for HMO subscribers and, in the case of primary care doctors, their referral of subscribers to specialists, is sufficiently low to leave the HMO a profit.

Why should you choose to contract with an HMO or an IPA or some other similar organization? Each contract will offer different opportunities and drawbacks, but some generalizations can be made.

The capitation payment arrangement provides a predictable income to the contracting doctor or group. Generally, a good advance estimate can be obtained of the number of HMO patients to be served in the practice. Therefore, under a typical arrangement, the provider can expect a more or less certain number of dollars at the beginning of each month under the contract. Thus income is assured.

The principal disadvantage of contracting with an HMO or IPA is the risk the provider assumes as to the health care needs of the subscribers. Since many HMOs require no or very low co-payments by subscribers, subscribers do not have any financial motivation to restrict their use of medical services. Thus, primary care doctors tell us of patients coming back several times for the same self-limiting illness. If the subscriber uses more than the predicted amount of doctor services, the doctor suffers. The assumption of this risk is a big step for a physician and one that

should be taken with great care, to avoid being overwhelmed by the "worried well." On the other hand, if the patients' demand for services is unexpectedly low, the doctor will benefit under most arrangements.

Some HMO or IPA contracts may offer a provider advantages other than the relatively predictable monthly income. A participating doctor may be able to (or may be required to) invest in the HMO or IPA. Such an investment may, of course, turn out to be profitable or otherwise, but the opportunity to invest is often desirable. If an HMO or IPA organization will be characteristic of medical practice in the future, a stake in one may give the doctor the chance to share in the profits to be realized in medicine other than as provider.

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***"Are you assuming  
liability for the  
acts of others?"***

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Participation in an HMO or IPA will invariably give the doctor or group an additional incentive to examine office procedures to insure that they are as efficient as possible. The capitation-payment or profit-sharing systems enable the doctor to benefit from practice efficiency even more than a fee-for-service system does. But it does not let inefficient providers survive very well.

### **PPOs**

Although the first preferred provider organization began operations in Denver only in 1980,<sup>2</sup> our experience indicates that the majority of alternative delivery system contracts offered to doctors today are with PPOs.

PPOs are groups of health care



**Detach and save  
for your reference**

providers—doctors and usually one or more hospitals—who contract with an insurer or directly with employers to provide services at an agreed-upon rate.<sup>3</sup> That rate is generally 10% to 20% lower than the provider's usual rate.

A patient enrolled in a PPO plan generally may choose to be treated by providers who have not contracted with the PPO, but that patient must then bear some of the costs of service. Thus the patient's choice of physician is protected, but while there is typically no co-payment for services of a participating provider, there may well be a large co-payment for services of a non-participating provider. So the incentive is clearly to use the "preferred" doctor.

One characteristic of PPOs is utilization review. Nearly all PPO contracts provide that a doctor who signs will submit to utilization review procedures, which usually are not specified in the contract. The procedures are intended to weed out from the PPO system expensive doctors. A doctor should learn in advance of signing a PPO contract as much as possible about how contracts of over-utilizers are affected.

Generally speaking, what advantages and disadvantages are there for a doctor or group in contracting with a PPO?

A principal advantage is easy, economic access to new patients,

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***"A 'hold-harmless'  
provision may be  
costly for you."***

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generally without sacrificing relationships with present patients. The doctor should first compare the amount of the discount provided for in the contract with his or her usual fee, less the cost of other means of increasing and maintaining his or her patient base. Then consider whether new competition may be willing to work for less to acquire the patient base. The choice here is difficult but must be faced.

Another principal advantage of the PPO is maintenance of the fee-for-service concept. Under an HMO arrangement, a doctor generally assumes the risk of heavy demand for his or her services, whether a capitation payment or a profit-sharing arrangement is used by the HMO. The provider does not become an insurer under a PPO contract. Thus the "loss" inherent in such arrangements is known, whereas it may not be in an HMO/ IPA set-up.

Another financial advantage of PPO contracts may be more rapid turn-around time for reimbursement. Some PPO contracts specify a time within which claims will be processed that is shorter than Blue Shield, Medicare and other insurance companies' turn-around time. A more rapid turn-around time would not, however, be an advantage vis-a-vis a capitation payment from an HMO or IPA.

Participation in a PPO can be a way for a doctor to compete more successfully with other alternative delivery systems that have taken root in the service area. Like HMO plans, PPO plans are a lower cost alternative to conventional health insurance. The doctor who has lost

patients to doctors who are under contract to HMOs might regain them or at least other patients as a PPO-participating provider.

Also like HMOs, PPOs give the contracting physician an incentive to examine the efficiency of his or her practice and office operations. With HMOs this incentive comes from the risk the doctor bears under the capitation payment or profit sharing system. Under the PPO, the incentive is provided by the narrowing of the provider's profit margin, which motivates the physician to see patients more efficiently and with less overhead.

On the other hand, participation in a PPO also has its disadvantages.

The utilization review procedures could result in a doctor's contract with the PPO being terminated—for good or possibly bad

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***"You may find  
the opportunity to  
invest desirable."***

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cause. To the extent that the doctor had structured his practice to serving the PPO patients, such a termination could be disastrous.

Another important disadvantage present in most cases is the reduction of reimbursements under traditional third-party insurance arrangements as a result of the contractual arrangement with the PPO. The discounted fees which the doctor agrees to accept under the PPO contract might reduce the doctor's "fee profile," resulting in a reduction of reimbursements from some third parties. For example, it is not clear how PPO arrangements affect Medicare profiles. Government regulations provide essentially for uniform fees to Medicare versus other patients, but PPO arrangements may result in lower Medicare revenues.

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***"You may be  
obligated beyond  
the contract's life."***

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This possible consequence of signing a PPO contract should be projected using the best available estimates and considered extremely carefully before the contract is signed.

The restrictions which the PPO contract may place upon a doctor's practice should also be carefully considered. Utilization review procedures would almost invariably include many such restrictions. Many contracts also require the doctor to be continuously on call for PPO patients or available for consultation with other PPO-contracting providers, which could present difficulties for solo practitioners. Sometimes the contract gives details of these responsibilities; sometimes it merely states that the doctor will perform such duties as the PPO decides. Contractual limitation of such open-ended power is clearly needed.

Frequently the contract will call for the doctor to maintain professional liability insurance at a specified level. This should not present a problem, but a contract which calls for the contracting doctor to "hold harmless" the PPO against any liability or claim that arises out of the services provided to PPO patients may cause the doctor to be liable for acts of others. If malpractice and other insurance policies exclude or limit coverage of liability the doctor assumes under a contract, the hold-harmless provision in the PPO contract may result in a substantial out-of-pocket expense to the doctor.

Almost any PPO contract will require the doctor to complete a great deal of paperwork throughout the term of the contract. An esti-

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***"Discounted fees  
could reduce  
your 'fee profile.' "***

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*"Easy access to  
new patients  
is an advantage."*

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mate of how burdensome this will be and what extra costs are involved should be made, although by itself this should not be a controlling factor.

An important part of any such contract is the termination provision. First, if the contract provides that either party may terminate it upon giving the other a specified number of days' notice (usually between 30 and 90), then a doctor will be able to terminate his or her responsibilities under the contract if the relationship is not working out. But remember that the PPO in turn can terminate the doctor's membership role at any time, if, for example, its management finds it can make a better deal with another doctor down the street. Second, the contract may place responsibilities on a doctor that would continue beyond the contract's termination date, such as continuing to treat PPO patients at the PPO rate for a length of time which may or may not be specified, or even requiring a doctor to continue to treat PPO patients at PPO rates without the option of declining to treat them.

Another potential disadvantage in a PPO contract is a restriction upon participating in other alternative health care delivery systems. The contract may forbid the doctor from joining another PPO or an HMO/IPA.

Finally, a weakness of most PPOs is their lack of a track record. A new organization may unexpectedly fold. A doctor should consider before signing the contract what the effect will be on his or her practice if the PPO is unable to meet its commitments.

## BROADER CONCERNS

When evaluating any contract with an HMO, an IPA or a PPO, many doctors will consider what the successful operation of the alternative delivery system might mean for the future of regional medical care delivery. A principal purpose of all these systems is, of course, to reduce the cost and increase the efficiency of the delivery of health care. Are these goals best met by adopting a capitation payment system for doctors, or by retaining the fee-for-service system and adding strict utilization review? Does making the doctor an insurer reduce the overall cost of health care delivery?

Answers to these questions are probably less attainable to the doctor than are answers to questions

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*"Your membership  
could be terminated  
at any time."*

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about how the particular contractual relationship might affect his or her practice, yet the broader questions must be of concern. If health care providers do not find acceptable approaches to cost containment, others, such as the government, will. Certainly, self-imposed formats will be better.

A doctor or group may be faced with the need to consider one or more contracts in a very short period of time. One group of doctors we have worked with received a proposed PPO contract from the local Blue Shield organization. The Blue Shield organization contract had to be "signed within two weeks," or the cost of joining its PPO would increase dramatically. Such high-pressure tactics should be strongly resisted, but care must be taken.

The accompanying brochure's explanation of what might be considered an anti-trust violation deserves careful attention. The result of these laws is that doctors should be very careful when engaging in any group discussion of alternative delivery system contracts; they should be prepared to consider these contracts individually.

## SUMMARY

The principal point of this article is that signing a contract with any alternative health care delivery system will have many effects on the physician and his practice—some apparent from a quick reading of the document, some only suggested by the document, some neither appearing in it nor suggested by it.

A contract is a binding legal document and should be understood as thoroughly as possible before it is signed. This article and the accompanying brochure, which is intended to be kept for reference, can help toward such an understanding, but the physician is well advised to obtain the advice of a lawyer or consultant before signing an alternative delivery system contract.

1. Reisser PC: We're still paying for our prepaid-care debacle. *Med Econ* Feb 6, 1984, p 97
2. Anon: Health care industry, business show increasing interest in PPO concept. *FAH Review*, July/August 1982, p 12
3. Ellwein LK, Gregg DD: InterStudy researchers trace progress of PPOs, provide insight into future growth. *FAH Review*, July/August 1982, p 20

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*"Review procedures  
almost always  
impose restrictions."*

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# Viruses and Arthritis

Duncan S. Owen, Jr., MD, *Richmond, Virginia*

In the past 25 years many reports have documented arthritis secondary to viral illnesses or vaccines. In some areas of the world these viral-induced arthritides can be extremely disabling; those in the United States are much milder. The exact mechanisms of synovitis are unknown, but deposition of immune complexes in the synovium has been documented in some.

**B**ACTERIAL INFECTIONS are always considered in the etiology of acute arthritis, but very little consideration is given to a viral etiology<sup>1</sup>. This review will attempt to emphasize the necessity of considering viral agents and will present data which possibly indicate viral etiologies for other diseases, e.g., rheumatoid arthritis.

## Arthritis With Viral Respiratory Syndromes

Viral syndromes consisting of fever, rash, coryza, headaches, myalgias and gastroenteritis occasionally may be associated with synovitis. Panush studied 11 patients with these symptoms; however, viral etiology was proved in only two: one with adenovirus and one with rubella. Musculoskeletal symptoms resolved in four weeks. Synovial effusions ranged from 2-60 ml; synovial fluid glucose

from 45-74 mg/dl; and total leukocyte count from 2,800-24,800/mm<sup>3</sup> with mainly polymorphonuclear leukocytes. Some synovial fluids had decreased complement levels<sup>2,3</sup>. Fortunately, these type of viral arthritides are usually transient and benign. However, one study in the United Kingdom reported three patients who developed seronegative arthritis in association with clinical and serological evidence of Coxsackie infection. One subsequently developed progressive erosive polyarthritis<sup>4</sup>.

It is interesting to note in various reviews about viruses and arthritis over the past few years that Lyme arthritis is included. Future papers will, of course, exclude Lyme disease as a viral infection because of the newly discovered etiology, a spirochete<sup>5</sup>.

## Hepatitis B Associated Arthritis

Over the past decade this has become a more frequently recognized entity notwithstanding it probably was first described in 1843. Approximately one-third of patients have musculoskeletal complaints. The arthritis, arthralgia and rash are probably secondary to circulating immune complexes, i.e., circulating aggregates of immune complexes, complement fixation and tissue injury. Arthritis and

From the Division of Immunology and Connective Tissue Diseases, Department of Medicine, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Owen at Box 647, MCV Station, Richmond VA 23298.

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dermatitis occur in the prodromal (pre-icteric) phase of the hepatitis. Joints involved are similar to those affected in rheumatoid arthritis. The affected joints are symmetrical in 75% of the cases. Laboratory tests usually are normal, with the exception, of course, of liver function tests, hepatitis B surface antigen (HBsAg) and serum complement. Synovial fluid complement may be depressed.

In chronic active hepatitis, 50% of patients have rheumatic complaints, usually arthralgia. The prevalence of musculoskeletal complaints with hepatitis A infection is unclear<sup>6,7</sup>.

### **Polyarteritis**

The relationship between polyarteritis and hepatitis B infection was first described in 1970. Today about one-third of patients with polyarteritis nodosa will be HBsAg positive. Synovial fluid complement is usually depressed. Vessel walls may contain antigen, complement and IgM. In addition, glomerulonephritis may be present<sup>8</sup>.

### **Essential Mixed Cryoglobulinemia-Purpura Syndrome (Meltzer-Franklin Syndrome)**

This entity was considered quite rare but is probably more common than is generally realized. Approximately one-half of these patients have anti-HBs and one-fourth have HBsAg. Some cryoprecipitates have particles consistent with hepatitis B virus. However, three-fourths of the cryoprecipitates contain either anti-HBs or HBsAg<sup>9</sup>.

### **Polymyalgia Rheumatica**

There have been some viral studies on this subject, but the data are inconclusive.

### **Rubella Arthritis**

Rubella arthritis is unusual in children, but in adults rubella may cause a severe transient arthritis in one-half of those affected. For some unknown reason about 90% of the adults with arthritis are females. Polyarthritis with morning stiffness is prominent. The arthritis coincides with or develops a few days after the rash. Arthritis lasts only a few days but arthralgias may persist for a month or more. Carpal and tarsal tunnel syndromes may occur. The virus has been cultured from joints. Serum complement may be depressed. To my knowledge there are no data on synovial fluid complement. Synovial fluid and synovial biopsies show 50% or more mononuclear cells<sup>1,6,7,10</sup>.

### **Post-Rubella Vaccination Arthritis**

Rubella virus was first isolated in 1962, and an

attenuated virus was subsequently used for vaccination. The strain used was called HPV-77 (high-passage virus 77), i.e., it had been passed 77 times in African green monkey kidney cell cultures. Other strains were passed in dog kidney, duck embryo and rabbit kidney cultures<sup>7,11</sup>.

The arthritis that follows attenuated live rubella vaccination is indistinguishable from that following natural rubella infection. Somewhere around 5%-10% of rubella-vaccinated children get arthritis; however, the older the person, the more likely they are to get arthritis, e.g., 50% of persons greater than 25 years old may get arthritis. Interestingly, there are post-arthritis arm and leg syndromes including one called "catcher's crouch".

### **Mumps Arthritis**

Arthritis associated with mumps rarely occurs. When observed, it is usually in male adults. Various joints, including cricoarytenoid, may be affected. Rheumatoid factor may be transiently positive. Since we know that mumps can cause an elevated serum lipase and since circulating lipase may produce a transient synovitis, it makes one wonder if this is a possible etiology.

### **Varicella Arthritis**

Arthritis is a rare complication of chicken pox. Examination of synovial fluid has shown 90% mononuclear cells. There is one case of pyarthrosis, apparently a consequence of bacterial infection of excoriated varicella skin lesions<sup>12,13</sup>.

### **Epidemic Erythema Infectiosum and Arthritis**

This is a mild erythematous illness of *presumed* viral etiology, mainly in children and occurring mainly in the winter and spring. Illness usually begins with fever, followed in 1-2 days by maculopapular malar erythematous eruption (the so-called "slapped cheek" appearance). There is also an evanescent rash in other areas. Headache, anorexia and gastrointestinal symptoms are commonly observed. Arthritis occurs in about 75% of adults but in less than 10% of children.

### **Smallpox Arthritis**

Osteomyelitis variolosa occurs in children less than 10 years old and has a striking predilection for symmetrical elbow involvement<sup>14</sup>. Presumably viral pustules form in the bones and joints. Elementary bodies have been identified in synovial fluid, but I am unaware of synovial fluid cultures being done.



### Smallpox Vaccination Arthritis

Acute monoarticular arthritis of the knee may occur 5-10 days after smallpox vaccination and persist approximately 10 days. Vaccinia virus has been cultured from synovial fluid. Examination of synovial fluid shows the glucose content to be about one-half that of the serum and synovial fluid leukocyte count approaches 50,000/mm<sup>3</sup> with 99% polymorphonuclear cells<sup>15</sup>.

### Infectious Mononucleosis and Arthritis

Arthralgias and arthritis probably occur more commonly than appreciated. Synovial fluid leukocytosis and neutrophilia mimic a septic process.

Most cases of infectious mononucleosis, probably all cases, are related to infections from the Epstein-Barr virus. There is some evidence that this virus may play a role in the pathogenesis of rheumatoid arthritis. This is based on the following observations:

1. Rheumatoid arthritis sera have precipitating antibodies to extracts of Epstein-Barr virus (EBV) Genome-positive B cell lines.
2. Rheumatoid arthritis sera have an increased frequency of an antibody to EBV cell lines detected by indirect immunofluorescence using anti-IgG. This antibody is called anti-RANA or rheumatoid arthritis-associated nuclear antigen.
3. Anti-RANA has been suggested as a marker for sero-positive and sero-negative rheumatoid arthritis.
4. Mononuclear cells of patients with rheumatoid arthritis show increased frequency and rapidity of both spontaneous transformation and transformation following EBV infection in vitro.
5. Antibody titers to the EBV structural antigens VCA (viral capsid antigen) and EA (early antigens) have been described as elevated in rheumatoid arthritis, although another study found normal values in rheumatoid arthritis patients.
6. Antibody titers to EB nuclear antigen (EBNA) have also been described as elevated or normal.
7. The elevated titers to IgG-VCA and EBNA do not appear to be secondary to the presence of rheumatoid factor<sup>16-22</sup>.

However, Silverman and Shumacher in December 1981, reported on 12 patients with rheumatoid arthritis seen within six weeks of onset of disease, and they did not have elevated titers of antibodies to VCA, early antigens, EBNA or RANA<sup>23</sup>. A year

later, a group in Tucson reported on nine patients with acute arthritis, four with chronic rheumatoid arthritis, and ten healthy adults<sup>24</sup>. Serial titers measured included antibodies to EBV antigens, group B Coxsackie virus, rubella virus, CMV and herpes simplex virus. Serological evidence of active EBV infection was found in four of the patients with acute arthritis, none with chronic arthritis, and one of the ten healthy adults.

### ECHO Virus Arthritis

This self-limited arthritis involves the wrist, fingers and knees and is usually associated with rash and fever. Clinically it can be mistaken as rubella arthritis.

### Adenovirus Arthritis

This type of arthritis can be caused by adenovirus type 7 infections. There is a case report of a 19-year-old military recruit who had a "viral illness" manifested by fever, chills, coryza, cough, pharyngitis, rash, myalgias and nuchal rigidity<sup>25</sup>. Arthritis of the knees resolved after seven days while the patient was receiving salicylates. Synovial fluid exam revealed a leukocyte count of 25,000/mm<sup>3</sup> with 99% polymorphonuclear leukocytes. Other cases have been reported which have showed cryoglobulinemia and immune complexes in both serum and synovial fluid specimens. IgG and C3 were found in cryoprecipitates, and lymphocytotoxic antibodies with specificities for membrane antigen determinants on B and T lymphocytes were found in two patients<sup>26</sup>.

### Arthritis Caused by Herpes Simplex Virus and Cytomegalovirus

Two cases of monoarticular arthritis caused by herpes simplex virus have been reported. The virus was isolated from synovial fluid in one patient and from other areas in the second; neither patient had known immunologic impairment<sup>27</sup>. In one patient arthritis lasted two weeks, but in the other it lasted four months.

A patient with a renal transplant developed synovitis of the right knee. Cytomegalovirus was isolated from synovial fluid, and electron microscopic studies revealed viral-like particles in the fluid<sup>27</sup>. The patient died several months later of fulminant pulmonary disease. Arthritis had not resolved.

### Arthritis in Association With Herpes Zoster

Arthritis associated with chicken pox has been previously discussed. Even though the same virus causes both chicken pox and shingles, arthritis with

the latter was not reported until 1979<sup>28</sup>. The arthritis is thought to be caused by direct inflammation of the synovium secondary to the virus spreading along the nerves from the dorsal roots. No sequelae have been reported, and the arthritis is short-lived. The arthritis, however, may be more common than is generally appreciated. Possibly the pain in the affected dermatome masks pain from a nearby joint<sup>29</sup>.

### Epidemic/Endemic Viral Arthritis

Chikungunya and o'nyonog-nyong fever, both epidemic in Africa and in Southern Australia, are Group A arboviruses, for which the vectors are mosquitos<sup>6-7</sup>.

The epidemic Ross River polyarthritis was described in Southern Australian adults in 1956. It has never actually been cultured from patients but has repeatedly been implicated by serological studies. Virus-like particles have been seen in electron microscope studies, and viral antigen has been found in synovial fluid.<sup>6</sup> Rash occurs in 50% of the patients. Arthritis is acute and severe. It is polyarticular but often is asymmetric.

Possibly viral in etiology are Navajo and New Guinea arthritis, and in Nigeria a condition called acute tropical polyarthritis. All are endemic and short-lived.

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# Grand Rounds: Pathogenesis of Infectious Mononucleosis

From the Department of Medicine  
University of Virginia School of Medicine  
Discussed by Richard P. Keeling, MD

## CASE PRESENTATION

**DR. MICHAEL E. WILLIAMS:** A 23-year-old female graduate student presented to the University of Virginia Student Health Service on 30 August 1982 complaining of swollen glands in her neck. She did not have sore throat, earache or fever but complained of pain from erupting wisdom teeth. On examination, she was afebrile. Non-tender gingivae surrounded erupting third molars. Pharyngeal examination revealed slight erythema and no exudate. The ears were normal. She had small ( $< 1.0$  cm), palpable lymph nodes in the right submandibular, left posterior cervical, and right and left anterior cervical chains. The heart and lungs were normal. Abdominal examination was normal, without splenomegaly. The skin was clear. Neurologic examination was normal.

The initial laboratory evaluation revealed hematocrit of 35% and WBC of  $4200/\text{mm}^3$  (35 granulocytes, 61 lymphocytes [20 atypical], 3 monocytes, and one eosinophil). A Monospot® test was nega-

tive. A throat culture done at the time of examination was subsequently reported negative.

A diagnosis of viral syndrome vs. infectious mononucleosis was made, and the patient was advised to rest, take fluids and aspirin, and avoid excessive alcohol intake.

She returned several times to the health service; Table 1 summarizes her course. About two weeks after her initial symptoms appeared, she began to complain of fatigue, and shortly thereafter she developed a faint maculopapular rash on the trunk and extremities. She became depressed and requested psychiatric assessment at the end of the third week of symptoms. At about the same time, her spleen was palpable and a Monospot test was positive. Her rash proved transient, her depression cleared without drug therapy, and her spleen tip regressed about eight weeks into the illness. Fatigue persisted for a total of five months, until January 1983.

Her atypical lymphocytosis peaked around the third week of illness; thereafter, she developed leukopenia (lowest WBC:  $3000/\text{mm}^3$ ). Atypical cells persisted for nearly four months, and her Monospot test remained positive for three. Her hematocrit returned spontaneously to normal after six months. Liver function tests done at the peak of her illness (weeks three and four) were normal, and a hepatitis B surface antigen was negative.

Presented on 6-9-83. Edited by Michael E. Williams, MD, and Marcia Day Finney. Address correspondence to Mrs. Finney at Box 466, Department of Medicine, University of Virginia School of Medicine, Charlottesville VA 22908.

## DISCUSSION

DR. KEELING: Many of you are primary care physicians who often see persons with infectious mononucleosis. As you studied the protocol, you may have noted that the woman whose case was presented is not the typical patient with this disease. Table 2 lists some features of infectious mononucleosis that we see typically in adolescents with the disease. Most patients have significant fever, severe sore throat, and/or lymphadenopathy. These symptoms were minimal or absent in the patient presented today. She did not have a palpable spleen at the outset of her illness but developed one later, and the same is true of a sense of fatigue, which is virtually the hallmark of the disease. She also had a rash, which is unusual in infectious mononucleosis, although the rash she had is the one typical of this illness—that is, a transient maculopapular rash on the trunk and proximal extremities. (It is worth noting that she was not given ampicillin; more than 90% of patients with infectious mononucleosis who are treated with this drug develop a rash similar to the one seen in this patient.) It is noteworthy that this patient was depressed. There is a high frequency of depressive episodes during the first few weeks of infectious mononucleosis. It is one of the most common sources of misdiagnosis, particularly when other features of the illness are atypical. For example, a college student seen recently in this hospital's emergency room for what was felt to be an acute adjustment reaction associated with examinations turned out on followup to have infectious mononucleosis. Most patients clear their depression spontaneously, as this woman did, and few require drug therapy for it. Although atypical at the time of presentation, this patient's illness in its later stages was more typical; the course summarized in Table 1 is a classic one.

Given her atypical presentation, how did this patient's physicians determine that she had infectious mononucleosis? The hematologic criteria generally accepted as diagnostic of something called the "mononucleosis syndrome" are as follows:

- Lymphocytosis  
    >5000 and/or >50% mononuclear cells
- Atypical lymphocytes  
    comprise >10% of white blood cells

This patient satisfied both criteria in that more than 50% of all her white blood cells were mononuclear cells and more than 10% of all her lymphocytes were atypical. These atypical lymphocytes (Figure 1) are fairly large, foamy, mononuclear cells which characteristically wrap themselves around red blood cell membranes. Laboratory findings which meet these two hematologic criteria reliably rule out other viral diseases that produce viral lymphocytes—so-called virocytes—in the blood, e.g., hepatitis or some adenovirus infections. However, such testing does not exclude certain other mononucleosis-like illnesses caused by agents such as cytomegalovirus. Arriving at the diagnosis of infectious mononucleosis in this patient was made more difficult by the fact that her heterophile (Monospot) test at the time she presented was negative.

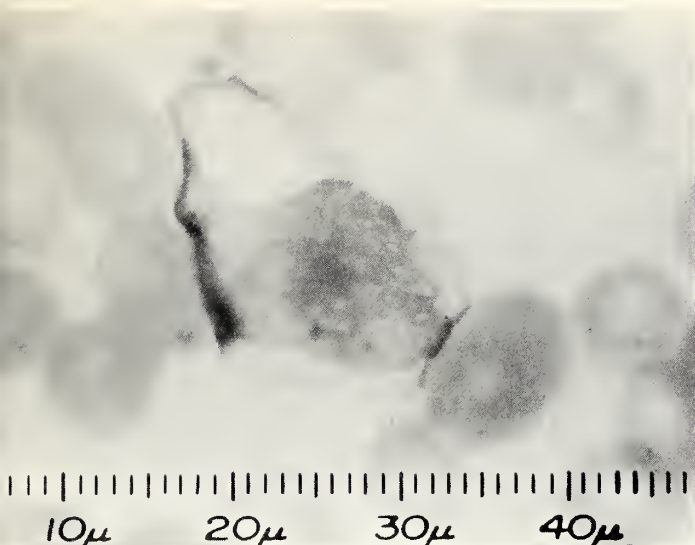
Table 2. Incidence of Features of Infectious Mononucleosis (1,329 cases/8 reports).

	Mean	Range
Fever	87.3%	67-98%
Cervical nodes	82.6	55-100
Sore throat	82.0	50-91
Splenomegaly	42.8	25-75
Palatine petechiae	31.0	13-50
Exudative pharyngitis	29.3	15-49
Hepatomegaly	10.6	5-24
Rash	8.1	1-15
Jaundice	5.4	0-11

Table 1. Summary of clinical and laboratory findings in a 23-year old with infectious mononucleosis.

Clinical and laboratory findings	Day of illness										
	5	11	14	18	20	39	62	89	113	144	178
Fever			±	±	±						
Pharyngitis	±										
Adenopathy	+	+	+	+	+	+	+	+	±	±	
Splenomegaly					+	+					
Rash			+	+							
Hematocrit	35	34	30	33	33	35	34	33	35	34	39
WBC	4.2	4.9	4.2	4.9	4.6	3.6	3.5	3.0	4.0	3.8	4.6
Lymphocytes (%)	61	54	55	54	56	57	55	58	26	25	23
Atypical lymphs (%)	20	20	21	28	38	18	19	8	4	2	3
Monospot	—	—	—	±	+	+	+	—	±	±	—
← - - - - - Depression - - - - - →											
← - - - - - Fatigue - - - - - →											





**Figure 1.** Atypical lymphocyte from the blood of a patient with infectious mononucleosis.

However, otherwise typical infectious mononucleosis is fairly frequently heterophile-negative. This happens often enough that one cannot safely rule out a diagnosis of infectious mononucleosis on the basis of a negative test alone. And, very often, a patient whose heterophile test is negative early in the course of illness later tests positive.

The Epstein-Barr virus (EBV) is a DNA virus of the herpesvirus group which has been established with reasonable certainty as the causative agent of typical infectious mononucleosis, the less typical mononucleosis syndrome represented by the case discussed today, and a variety of other disease entities. The virus was first associated with lymphoid diseases as early as 1964, when it was identified in cell culture lines from a patient with African Burkitt's lymphoma.<sup>1</sup> Its role in infectious mononucleosis was first hypothesized in 1968 by Drs. Werner and Gertrude Henle.<sup>2</sup> The evidence is as follows. First, only those people who do not have preexisting EBV antibody contract infectious mononucleosis. Second, acute infectious mononucleosis is associated with the appearance first of an IgM and then of an IgG antibody to EBV. Third, only those persons who are seropositive for the virus have EBV-containing lymphoblastoid cell lines in the peripheral blood. In patients with acute infectious mononucleosis or in those recovering from the disease, one can detect EBV particles in lymphocytes in the peripheral blood and in cell-free washes of the oropharynx; such evidence of the virus's presence is never found in seronegative persons. In addition, there is evidence that EBV can be transmitted to seronegative persons by

transfusion of blood products from persons with acute infectious mononucleosis.

The EBV is associated with a broad spectrum of disease. There is, of course, infectious mononucleosis. There are some cancers, which I will mention later. There is an important, and common, subclinical illness which occurs principally in children and young adults and is often characterized only by seroconversion. There is also a progressive, devastating, nearly always fatal lymphoid disorder that affects males who have a congenital immunodeficiency; this illness looks like infectious mononucleosis but is not a self-limited disease process.

Epidemiologic studies in recent years have shed some light on why and how EBV produces different clinical manifestations. Infectious mononucleosis is not a very contagious disease. For instance, when a college student living in a dormitory is diagnosed as having infectious mononucleosis, only very rarely does anyone else on the hall get the disease. The EBV seems to behave much like the polio virus in that it is most commonly transmitted among children by fecal-oral contact. In children, it produces a very mild disease which bears little resemblance to infectious mononucleosis; in fact, this illness is often marked only by seroconversion to EBV. It was once thought that such childhood infections occurred only among populations in Africa, but recent studies of children in the United States have confirmed that seroconversion to EBV takes place commonly here and that the incidence of definable illness accompanying seroconversion in children is very low. Depending on socioeconomic conditions which facilitate or hinder fecal-oral contact and so promote or discourage transmission of EBV, a variable percentage of the population develops subclinical EBV infection at an early age and thereby acquires protective immunity to the virus. In Africa, more than 90% of children are seropositive for EBV by age 5, and nearly 100% have immunity by age 10.

Where sanitary conditions are better and fecal-oral transmission of virus is less easily accomplished, particularly in affluent, Western societies, a smaller percentage of the population becomes seropositive for EBV during early childhood. In the United States, as much as 40% of the college-age population (18–22 years of age) is still seronegative and susceptible to the virus, and a significant portion of this group comes from the higher socioeconomic strata. In adolescence and the college years, expression of primary EBV infection frequently is not subclinical. Rather, it takes the form of typical infectious mononucleosis. Epidemiologically

speaking, then, typical mononucleosis is, to some degree, a disease of the affluent.

Persons who remain seronegative throughout childhood, adolescence, and their college years and contract EBV infection when they are over 20 years of age usually develop an atypical form of mononucleosis. The illness may resemble that of the patient presented today, and its presentation may be similar to that of a typical primary infection with cytomegalovirus. Compared with teenagers who have atypical infectious mononucleosis, young adults with EBV infection may experience lesser degrees of pharyngitis, lymphadenopathy, and liver function abnormalities but a greater sense of fatigue.

In all populations, including that of the United States, virtually 100% of persons are seropositive for EBV by 40 years of age. Primary infections with EBV are almost unknown in individuals over 40.

Even within susceptible populations, the rate of EBV transmission is relatively low. Of susceptible close contacts of persons who have acute infectious mononucleosis and are excreting virus, only 15% actually develop any form of EBV infection; of those who contract infection and seroconvert, only a minority develops typical infectious mononucleosis. The best study documenting this was carried out at West Point, where one year's entire entering freshman class was followed for four years.<sup>3</sup> About one-third of the students were seronegative on arrival as freshmen; 20% of them converted to seropositive each year during their four years at West Point, but, of those who converted each year, only one-fourth had a clinical illness which could be identified as typical infectious mononucleosis.

How, then, is EBV infection spread? There is authoritative medical evidence now to substantiate what college students have always suspected, that kissing is the culprit. Cell-free washes of the oropharynx of patients with acute infectious mononucleosis reliably yield infective EBV particles. One can also obtain viral particles by cannulating the parotid ducts of persons with acute infectious mononucleosis. The virus persists continuously in the oropharynx of persons recovering from acute infectious mononucleosis for up to 18 months and appear in that site sporadically thereafter; at any given time, 10–15% of seropositive individuals in the healthy population—and up to 50% of immunosuppressed persons—are shedding EBV from the oropharynx. What this means is that the reservoir for acquisition of infectious mononucleosis or other EBV infection consists of most of the general population and is not limited to persons with acute

EBV infection of one kind or another. It is therefore exceedingly difficult to identify case-to-case contacts for EBV infections.

A variant mode of transmission of EBV infection is via transfusion of infected blood products. This is a very rare occurrence, because viremia lasts for only a day or two after infection is acquired and because disease develops only in seronegative persons receiving the live virus in transfused blood. On the basis of studies of EBV transmission by transfusion, the incubation period for EBV infection has been estimated to be four to eight weeks.

When the virus enters a susceptible host, it infects first some cells in the oropharynx, most likely the B lymphocytes in Waldeyer's tonsillar ring. The list of cell types having EBV receptors is very short—only B lymphocytes and a small proportion of non-B, non-T lymphocytes are so equipped. Once in the B cell, the virus acts as a polyclonal B-lymphocyte activator, causing unrestricted polyclonal proliferation of B cells and, eventually, production of antibody and consequent hypergammaglobulinemia, a dominant feature of the first few weeks of EBV infection. EBV-infected lymphocytes meet one of three fates. They may be lysed, in which case intact EBV and viral antigens are released. Or, cells may become latently infected so that the replicative cycle of the virus stops early and only certain viral antigens are expressed, notably the lymphocyte-determined membrane antigen, which is detectable by antibody and does not further affect the cells. Finally, cells may be transformed, establishing vampire-like relationships with the virus and thereby becoming immortal. These "undead," EBV-transformed lymphocytes will reproduce themselves *ad infinitum* in cell culture.

The virus then spreads throughout the body, either by means of actual viremia or by a "B-lymphocytemia," in which the infected B cells travel along the blood stream to other sites. Intact virus reproduces itself and is shed from the oropharynx as early as eight days after entering the body, long before any symptoms appear; thus, infection is spread before the patient is even aware that he is ill.

The developing infection is a virological nightmare, with EBV-infected, polyclonally activated B cells circulating, intact virus being produced, more and more B cells becoming infected and stimulated, more and more B cells being produced, and more and more antibodies being manufactured. Questions arise: Why doesn't EBV infection uniformly become a B-cell lymphoma? Why doesn't B-cell acute lymphocytic leukemia always arise from



**Table 3. EBV-Specific Antibody Patterns.**

Susceptible person: no antibody
Patient with acute infectious mononucleosis:
+ IgM, IgG viral capsid antibodies
+ early antibody, diffuse staining pattern (EA-D)
+ early antibody, restricted staining pattern (EA-R), appearing after EA-D
Patient convalescing from infectious mononucleosis:
+ IgG viral capsid antibody
+ IgM viral capsid antibody
rising EBNA

acute infectious mononucleosis? What is the difference between infectious mononucleosis and cancer? The answer to all such queries has to do with the host's immune response—both humoral and cell-mediated—to the EBV and to the infected B cells.

Antibody production in infectious mononucleosis is of two types. There are specific antibodies which limit the proliferation of virus and B cells and so limit the spread of infection in the first few weeks and which protect the host for years against reinfection with EBV. There are also some non-specific antibodies which, although they are epiphenomena of the disease and have nothing to do with the EBV itself, serve as markers of EBV infection.

The earliest detectable EBV-determined antigen is called the Epstein-Barr nuclear antigen (EBNA); this is accepted as a marker of cellular transformation by EBV. Antibody to EBNA develops during the late acute or early convalescent phase of infectious mononucleosis and can be interpreted as evidence of previous EBV infection. Some infected B cells, those characterized as producers, manufacture viral particles that create viral antigens which in turn elicit an antibody response; these B cells also express other EBV-determined cell antigens, including transient IgM and permanent IgG antibodies to the capsid of the virus, transient early antigens which develop in the first week of infection and elicit antibody in about 80% of patients, and an apparently virus-neutralizing permanent antibody to a lymphocyte-determined membrane antigen.

Table 3 shows how one can use immunological tests to determine the EBV exposure status of a given patient. Susceptible persons, obviously, will not have antibody to any of the abovementioned antigens. A person with ongoing acute infectious mononucleosis will have a positive IgM viral capsid antibody and, possibly, a low titer of an IgG viral capsid antibody. He or she may also have early antibody to different types of early antigens. A person beginning to recover from infectious mono-

nucleosis will have a high titer of antibody to the IgG viral capsid antigen and will lose the IgM viral capsid antibody; he or she will also have a rising titer of antibody to EBNA. Most commercially available kits for testing for EBV antibodies detect the IgG viral capsid antibody; that is, they detect immunity to the virus, not acute infection, and therefore determine only whether the patient has ever had EBV infection. In order to detect current, active infection and to specify its etiology, one must test for a positive IgM antibody; such a test is available but is quite costly. A rising EBNA titer will also indicate active infection. However, for clinical purposes, such testing is rarely needed. It is useful principally when one is caring for patients with immunodeficiencies and unusual illnesses or for those with heterophile-negative infectious mononucleosis for whom the etiology of disease must be established.

In addition to the specific EBV antibodies, there are certain other non-EBV-specific antibodies generated during acute EBV infection. The most famous of these are the heterophile antibodies, which are present in up to 90% of patients with infectious mononucleosis at some point during the illness. The role of these antibodies in the pathogenesis of or recovery from infectious mononucleosis is unclear, but detection of them in a titer of more than 40 in a patient with a compatible clinical presentation is strong evidence for infectious mononucleosis. In testing for heterophile antibodies, the classic tube differential heterophile test and the Monospot test are virtually interchangeable, and there is a concordance rate of testing of better than 97% among the slide test, the Monospot test, the Monosticon® test, and the differential heterophile. Heterophile testing in infectious mononucleosis yields results of which the following are typical: 75–90% of persons with infectious mononucleosis due to the EBV are heterophile-positive, and 15–45% of persons shown to be heterophile-negative have disease caused by EBV. Because the EBV causes both heterophile-positive and heterophile-negative infectious mononucleosis, a positive heterophile test specifies the etiology as EBV but is not diagnostic. There is no relation whatever between the positivity or negativity of a Monospot test or a heterophile titer and the symptomatology or severity of a patient's clinical illness. The heterophile test usually turns from positive to negative in the fifth or sixth month after infection, as it did for the patient discussed today.

Among the other antibodies seen in infectious mononucleosis are anti-i antibody, antiplatelet antibody, antigranulocyte antibody, antinuclear factor,

rheumatoid factor, VDRL and smooth muscle antibody. Most of these have little or no pathogenic significance, although one that may have clinical relevance is the anti-i antibody to a red-cell i antigen. A cold agglutinin, this antibody is found in about 8% of persons with infectious mononucleosis; in a very small percentage of the already small number of persons carrying i antigen on red cells, it produces a cold-agglutinin hemolytic anemia. The only other of these antibodies which can cause disease of much consequence is the antiplatelet antibody, which occasionally produces an illness resembling idiopathic thrombocytopenic purpura as either the sole presentation or a convalescent phenomenon of infectious mononucleosis. There is also a granulocyte autoantibody which may be responsible for the granulocytopenia observed in some persons with EBV infections.

To return to the EBV-infected B cells—what about the host's cell-mediated response to them? What are the T cells doing? While the first week after infection is characterized by polyclonal hypergammaglobulinemia and extensive proliferation of B cells, the second week is marked by a dramatic reduction in immunoglobulin levels, a drop in the number of antibody-secreting cells, and a decrease in the number of B cells in the peripheral blood; all of these developments correlate with an enormous increase in suppressor/cytotoxic T-cell subgroup activity. Specific cytotoxic/effector T cells direct their activity at the EBV-transformed B lymphocytes, precipitating a phenomenon which has been termed the "war of the lymphocytes." It is this conflict between the infected B cells and the cytotoxic T cells that is largely responsible for the clinical manifestations of infectious mononucleosis.

The T-cell reactivity is specific and HLA-restricted and develops only in persons who are seropositive for EBV infection. Only rarely is it seen in the first week of illness, but 50% of patients have it by the third week and it is present in virtually all patients by the fourth or fifth week. The cytotoxic cell population peaks between the fourth and ninth weeks of illness, and this event correlates with the beginning of a moderation in the clinical symptomatology and with development of high levels of specific antibodies to EBV.

The "war of the lymphocytes" produces an intense lymphoid hyperplasia which manifests itself clinically as adenopathy and hepatosplenomegaly; sometimes, patients develop myocarditis, pericarditis, or polyneuritis. Early in the disease course, the cells in the lymph nodes and liver are a mixture of B and T cells, but, late in the disease, the cell popula-

tion at these sites consists almost exclusively of T cells. The T-cell response is most easily documented by the detection of atypical lymphocytes in the peripheral blood. The vast majority of these circulating atypical lymphocytes are T cells and, specifically, cytotoxic/effector T cells for EBV-bearing B cells. A direct correlation exists between the number of circulating atypical lymphocytes and the amount of in vitro cytotoxicity demonstrable in a patient.

Over time, the T cells gradually bring the B cell proliferation under control, the number of circulating atypical lymphocytes drops, and there is a reduction in cytotoxicity; eventually, the cytotoxic T cells disappear altogether from the peripheral blood. They are replaced, however, by a permanent population of memory T cells. In addition, there is antibody-dependent cellular cytotoxicity involved in protection against infected B cells and, in the initial weeks of illness, natural killer cell activity directed against lytic producer cell groups of EBV-infected B lymphocytes. Much of the host defense in the acute period of infectious mononucleosis is the result of non-specific natural killer cell activity, while control of the middle and convalescent phases of the disease is the responsibility of the specific T cells.

Following an EBV infection such as infectious mononucleosis, a lifetime EBV carrier state develops, accounting for periodic shedding of the virus in the oropharynx, even when the host is asymptomatic. This implies the existence of a sort of immunological sanctuary in which, at least occasionally, intact virus is produced by replicative cycles. This sanctuary is most likely located in the salivary glands. In addition, after infectious mononucleosis a person has small numbers of immortal, transformed B lymphocytes circulating in the peripheral blood for the rest of his life; these cells, if planted in tissue culture, will yield continuous lymphoblastoid cell lines which will be permanently EBNA-positive.

Persistent viral activity is matched by the presence in the body of at least two deterrent forces. One is the persistence throughout the host's life of relatively high levels of EBV-specific antibody. Second, there is chronic specific T-cell activation which develops after clinical or subclinical disease and is characterized by long-term surveillance of the EBV carrier state. One can find in the blood of most of us EBV-specific memory T cells with cytotoxic potential. When these cells are placed in tissue culture with virus-infected lymphocytes, they become cytotoxic and inhibit the outgrowth of



EBV-transformed cell lines. This probably constitutes the host's first line of defense against the future reactivation of infection. It appears that such specific cytotoxic T cells are constantly eliminating in the host latently infected B cells.

Immunologic control of EBV is actually a rather delicate balance, and the potential for its disruption is more than a theoretical possibility. There are, in fact, some situations in which control of EBV infection is inadequate. These are principally of interest because they involve a number of EBNA-positive tumors thought to be related etiologically to the EBV, including nasopharyngeal carcinoma, African Burkitt's lymphoma, some Burkitt's lymphoma in Americans (mainly in persons with acquired immune deficiency syndrome [AIDS]), B-cell lymphoma arising in some patients on immunosuppressive regimens, and certain plasmacytomas and B-cell lymphomas occurring in patients with another immunodeficiency syndrome.

It appears that patients on immunosuppressive therapy are at a higher risk for reactivation of EBV infection and for acquisition of EBV-related tumors. Most studies of this phenomenon have been done with renal transplant recipients on immunosuppressive drugs. Virtually all of these patients have decreased EBV-specific T cell function; in many, T cell responsiveness is profoundly depressed and remains so for as long as 15 years after renal transplantation. Also, the rate of EBV shedding in the oropharynx in renal transplant patients increases in direct proportion to the degree of immunosuppression. For example, the rate of EBV excretion rises when a patient is given antilymphocyte globulin or when he is placed on a more prolonged drug-induced immunosuppressive regimen. Fully half of the renal transplant patients given immunosuppressive drugs become persistent EBV excretors.

The response of immunosuppressed patients to EBV is interesting. We see in this population an increased number of EBV-related tumors and a wide range of disorders—from B cell hyperplasia to B cell monoclonal lymphomas—in which the cells are EBNA-positive and in which the EBV genome is demonstrable. Furthermore, in some of these patients, an immunoblastic lymphadenopathy progresses to immunoblastic sarcoma.

The most dramatic breakdown in the control of EBV occurs in persons with the congenital immunodeficiency known as the X-linked lymphoproliferative syndrome (XLPS). First described in 1979, this syndrome is characterized by impairment of the host's immune response to EBV which allows for

development of overwhelming, fatal infectious mononucleosis or EBV-related neoplasia.<sup>4</sup> The disorder is X-linked, occurring only in males, and has been seen in boys and young men from ages 6 months to 22 years. Most have had some other sort of mild immunodeficiency at the time they contracted EBV infection. Described in more than 100 patients to date, XLPS causes a devastating illness; more than 80% of its victims die within three years of diagnosis.

In these patients, a defective T cell response results in poor and insufficient control of the B cell proliferation triggered by the EBV. The B cells accumulate, causing organ infarction and multiple organ failure; most patients die of hepatic necrosis. The syndrome has several variants which are dependent on the nature and degree of the immunological defect. Persons with the most severe defect contract overwhelming infectious mononucleosis. This is the most common form of XLPS and occurs at an average age of 6½; the average survival time after the onset of symptoms is less than 50 days. EBV is detectable in cell genomes. Autopsy reveals extensive hepatic necrosis, with B cells everywhere and no sign of a T cell response. Some of these patients also develop aplastic anemia. Patients with less severe defects, about 25% of those persons with XLPS, develop B cell lymphomas—Burkitt's lymphoma or plasmacytoid or immunoblastic lymphomas. Treatment of immunodeficient patients with acyclovir has not been encouraging, and, of the four patients so treated to date, none has derived any benefit.<sup>5</sup>

XLPS is not the only known EBV-related immunological defect. Among patients with AIDS, for example, a small percentage develop B cell lymphomas, either burkittoid or undifferentiated in type. Most AIDS patients with such lymphomas test positive for EBNA and the EBV genome is demonstrable by molecular hybridization. Most AIDS patients also experience increased shedding of EBV in the oropharynx. Could this mean that EBV is the cause of AIDS? This seems unlikely for many complex reasons; cytomegalovirus at present seems a more likely candidate.

As this review has shown, EBV infection expressed as infectious mononucleosis is a model of self-limited lymphoproliferation. The EBV might better be described as a mitogen than as an oncogen<sup>6</sup> in that it elicits in the susceptible host a complex system of breaches in immunologic control which produce relatively predictable—and usually fairly benign—consequences.

*References on page 355*

# Hypereosinophilic Syndrome: A Review

Lawrence B. Schwartz, MD, *Richmond, Virginia*

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Prolonged eosinophilia causing organ system complications results in hypereosinophilic syndrome, with involvement of cardiac and central nervous systems producing the more severe disease. Initial treatment includes steroids; further therapy may utilize cytotoxic drugs and surgical intervention. Aggressive management has contributed to a current survival rate at five years of 80%.

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**T**HE hypereosinophilic syndrome (HES) can be defined as eosinophilia 1) that is equal to or greater than 1500 eosinophils/mm<sup>3</sup>; 2) persists for at least six months; 3) results in organ system dysfunction; 4) and absence of a recognized cause for the eosinophilia. The eosinophilia should be primary, i.e., recognized causes of secondary eosinophilia (Table 1), such as allergy or parasite infestation, must be excluded. The many individuals who have eosinophilia without organ system sequelae are not considered to be a subgroup because it is the combination of persistence of eosino-

From the Department of Internal Medicine, Division of Immunology/Connective Tissue Diseases, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to Dr. Schwartz at Box 204, MCV Station, Richmond VA 23298.

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philia with organ system dysfunction that best defines this syndrome.

Although the hypereosinophilic syndrome was originally defined in the context of primary eosinophilia, it is now recognized that the same organ system complications may arise when eosinophilia occurs secondary to a defined condition. However, because treatment of the primary condition is directed at reduction of the eosinophil concentration and treatment of the secondary forms is directed at the underlying disease, distinguishing primary from secondary hypereosinophilia is very important.

In this review our current understanding of the special components of eosinophils and their postulated clinical correlates will be reviewed in the context of the hypereosinophilic syndrome.

## Production/Distribution of Eosinophils

Eosinophil concentrations in blood are normally less than 500/mm<sup>3</sup>, but a diurnal variation in the concentration of eosinophils in circulation causes these concentrations to vary about two-fold, appar-



ently in a reciprocal relationship to plasma cortisol levels, i.e., eosinophil counts peak in the evening and bottom out in the morning.<sup>3</sup> Furthermore, eosinophil counts in the blood may not reflect eosinophil concentrations in tissues, where, as is well documented in animals,<sup>4</sup> greater than 99% of mature eosinophils are thought to reside. A complex array of factors must be called into play for eosinophils to develop from uncommitted stem cells in the bone marrow and migrate into tissues.<sup>5</sup> These factors include colony-stimulating factor (CSF), which causes stem cells to become committed to an eosinophil lineage; eosinophilopoietic factor (EPP), a T-cell-derived factor that accelerates proliferation and maturation of eosinophils in the bone marrow; eosinophil-release factor (ERF), which releases eosinophils from the bone marrow into the circulation; putative activities that promote eosinophil adherence to vascular endothelia and penetration into tissues; and chemotactic factors that direct the migration of tissue eosinophils to a specific site and concomitantly cause the appearance of increased numbers of receptors for C3b and C4 on the eosinophil surface. Numbers of receptors for IgG remain unchanged.

Eosinophils encountering a noxious substance are capable of mounting a phagocytic response that results in the union of the ingested material and eosinophil granules to form a phagolysosome.<sup>9</sup> If this perturbing substance is too large, such as a schistosomule, a secretory response occurs and the contents of specific granules are deposited onto the schistosomule in the extracellular space.<sup>10</sup> The fate of lysosomal enzymes and other components residing in the small granules is less clear, though release of arylsulfatase and beta-glucuronidase occurs when human eosinophils are exposed to opsonized zymosan and to C5a. Thus, directed migration of eosinophils results in an enhanced capacity to opsonize antibody and complement-coated invading organisms or noxious substances.

### Morphology

Eosinophils can be recognized by light microscopy because of their bilobed eccentrically-placed nucleus and acidophilic peroxidase-rich cytoplasmic granules. By electron microscopy mature eosinophils can be seen to have at least two granule subtypes. The *specific* granules are ellipsoid ( $0.5\text{--}1.5\text{ }\mu\text{m} \times 0.3\text{--}1.0\text{ }\mu\text{m}$ ) and have a rectangular electron-dense core surrounded by a less electron-dense matrix that is characteristic of eosinophils.<sup>7</sup> Another granule subtype is the *small* granule. These granules are found only in mature eosinophils and

**Table 1. Recognized causes of secondary eosinophilia.**

Allergy
tropical eosinophilia
asthma
rhinitis
bronchopulmonary aspergillosis
drug reactions
Infection
parasitism
tissue invasive helminths
protozoa
pneumocystis carinii
bacteria
scarlet fever
tuberculosis
chlamydia
mycoses
coccidioidomycosis
chronic mucocutaneous candidiasis
Dermopathy
atopic dermatitis
dermatitis herpetiformis
eosinophilic fasciitis
exfoliative dermatitis
Rheumatism
necrotizing vasculitis
allergic angiitis
Churg-Strauss syndrome
rheumatoid arthritis
systemic lupus erythematosus
scleroderma
dermatomyositis
Hepatopathy
cholestatic hepatitis
Cardiopathy
serous myocarditis
Neoplasia
lymphoma
Hodgkins disease
mycosis fungoides
leukemia
acute lymphoblastic leukemia
Sezary syndrome
solid tumors
Immunodeficiencies
cyclic neutropenia
Wiskott-Aldrich syndrome
hyper-IgE syndrome
IgA deficiency
graft versus host disease
Endocrine
Addison's disease
pituitary hypofunction
Nephropathy
interstitial nephritis

are spherical, variable in diameter (0.05-0.5  $\mu\text{m}$ ), and amorphous.<sup>8</sup>

### Mediators

The chemical composition of granule and non-granule compartments of the eosinophil have in part been defined and indicate special functional attributes (Table 2). Approximately 50% of the secretory granule protein on a weight basis is composed of a 9400 mw protein, termed major basic protein

Table 2. Mediators of Eosinophils.

Mediator	Activity
Preformed	
major basic protein	cidal to cells and microorganisms
eosinophil cationic protein	thrombogenic
peroxidase	antimicrobial
histaminase	degrades histamine
arylsulfatase	removes sulfate from aromatic residues
phospholipase D	removes phosphatidyl moiety from phospholipids
lysophospholipase	removes fatty acid group from lysophospholipids
Newly Generated	
prostaglandin E	bronchodilate, vasodilate
leukotriene C	bronchoconstrict, vasodilate, increase vascular permeability, decrease cardiac contractility

(MBP), that is rich in arginine and cysteine residues.<sup>11</sup> The protein has cytotoxic properties against endothelial and epithelial cells *in vitro*<sup>12</sup> at concentrations that are known to occur in sera of patients with eosinophilia<sup>13</sup> and in sputa of atopic asthmatics.<sup>14</sup> In the former patients MBP has been postulated as a means by which damage to the endocardium may occur, and in the latter group of asthmatic patients it may impair the integrity of the epithelial lining of the respiratory tissue. Although MBP is predominantly located in eosinophils, it has also been detected in basophils.<sup>15</sup> Another component of the specific granule is eosinophil cationic protein (ECP), a 21,000 mw protein that has the capacity to enhance activation of Hageman factor (Factor XII) and plasminogen and may help to explain the thrombogenic sequelae sometimes seen in the hypereosinophilic syndrome.<sup>16</sup> Both MBP and ECP are acidophilic and account for the staining characteristics of eosinophils with acid dyes.

Eosinophils stain more intensely for peroxidase than either neutrophils or monocytes.<sup>17</sup> Eosinophil peroxidase is in the matrix of the specific granule. The enzyme is immunologically, structurally, and genetically different from myeloperoxidase of neutrophils. It is insensitive to inhibition by cyanide; utilizes iodide and not chloride; inactivates sulfido-peptide leukotrienes (slow reacting substances) by oxidation; and in the presence of iodide and H<sub>2</sub>O<sub>2</sub> is cidal for a variety of cells and microorganisms. The combined activities of eosinophil peroxidase and MBP may be important for the helminthotoxicity of eosinophils.

Small granules contain a variety of lysosomal acid hydrolases, including beta-glucuronidase and

arylsulfatase. Both of these enzymes are released when human eosinophils are exposed to opsonized zymosan and to C5a. Arylsulfatase at one time was thought to inactivate slow-reacting substance of anaphylaxis (SRS),<sup>18</sup> but with the availability of purified enzyme and pure, chemically defined, slow-reacting substance, aryl sulfatase has been shown not to inactivate SRS.<sup>19</sup> Eosinophil histaminase, though not yet localized to a particular subcellular compartment, can inactivate histamine released by mast cells and basophils.<sup>20</sup> However, rapid metabolism of histamine by monamine oxidase and histamine-N-methyl transferase in plasma and tissues probably overshadow the capacity of eosinophils to degrade this metabolite under most conditions. Phospholipase D removes the phosphatidyl moiety from lysophospholipids, e.g., choline from phosphatidyl choline. Although at one time phospholipase D was thought to inactivate platelet-activating factor (PAF), the moiety inactivated turned out to be a lysophospholipid that lysed platelets, whereas noncytotoxic PAF was unaffected.<sup>21,22</sup>

Lysophospholipase is an enzyme present in eosinophil membranes that removes the fatty acid from the C-1 position of lysophospholipids. It appears to account for 5%-10% of total cell protein. This enzyme crystallizes to form the characteristic Charcot Leyden crystal that has been so closely associated with increased numbers of eosinophils. Charcot Leyden crystals formed *in vivo* and isolated and examined *in vitro* contain a single protein component, the lysophospholipase.<sup>24</sup>

In addition to protein components, eosinophils have the capacity to generate products of arachidonic acid. These include leukotrienes, via the lipoxygenase pathway,<sup>25</sup> and prostaglandins, via the cyclooxygenase pathway.<sup>26</sup> The potent activities of leukotrienes and prostaglandins have been reviewed elsewhere.<sup>27,28</sup>

### Clinical Presentation

Patients with hypereosinophilic syndrome are usually male, 20 to 50 years of age, and suffer a greater than 50% mortality by one year when untreated.<sup>1,2,6,29</sup> Most likely, HES represents the end result of a heterogeneous group of diseases with the common denominator of eosinophilia and tissue damage. Though eosinophils residing in the bone marrow of patients with the syndrome appear normal by light microscopy, peripheral blood eosinophils may appear vacuolated, suggesting prior degranulation and release of granule contents.<sup>1,30</sup> The complications of HES appear not to be related



solely to the concentration of eosinophils, but perhaps more to the extent of degranulation. Prolonged eosinophilia may never cause organ system dysfunction in some individuals and therefore by itself requires no treatment.

Patients with hypereosinophilic syndrome typically present with non-specific constitutional symptoms. Weakness, fatigue, cough, dyspnea, myalgias, angioedema, rash, fever or rhinitis were listed as presenting symptoms in about 90% of patients, whereas in the remainder eosinophilia was found by routine laboratory tests, often during investigation of an apparently unrelated medical problem.<sup>1</sup> Night sweats are also common.<sup>2,6,29</sup> Although any organ system may be involved, cardiovascular lesions are a characteristic complication of HES, being present in about 84% of this population,<sup>2</sup> and are associated with a worse prognosis. Involvement of the left heart chamber often predominates.<sup>1,31</sup> Endothelial cell damage and necrosis of the ventricular wall and intramural coronary endothelia occur early, and thrombi commonly develop over the damaged area. Embolic sequelae may occur. Later there is fibrosis that may involve the posterior leaflets of the mitral or tricuspid valves. Mitral regurgitation, pulmonary edema, and a third heart sound are signs of associated left ventricular dysfunction. Tricuspid regurgitation, hepatomegaly, peripheral edema, and ascites are signs of right ventricular dysfunction and usually indicate that biventricular damage to the heart has occurred. In patients with prolonged eosinophilia, two-dimensional echocardiography and electrocardiography may detect a thickened posterior mitral valve leaflet attached to a thickened left ventricular posterior wall and endomyocardial echos reflecting endomyocardial fibrosis. Similarly, development of ST depression and T-wave inversion on the EKG may indicate cardiac involvement. Invasive tests, such as angiography or endomyocardial biopsy, may be required when the diagnosis is in doubt or the extent of dysfunction needs a more objective evaluation. Angiography may show apical filling defects and endomyocardial biopsies may show acute necrosis, thrombosis, and/or tissue eosinophils early in the disease and a thickened fibrotic endocardium at a later stage. Endomyocardial fibrosis, a late sequela of the hypereosinophilic syndrome, is often found at postmortem examination.

Hematopoietic involvement is found in over 90% of individuals.<sup>2</sup> Mild anemia is associated with normal levels of iron and total iron binding capacity and a hypercellular bone marrow, with a 5-15/1 myeloid/erythroid ratio and 25-75% of the marrow

cells being eosinophils. When myeloblasts are found in peripheral blood or the total white blood cell concentration is greater than 100,000 per mm<sup>3</sup>, the prognosis is grave, with only about 25% alive at one year.

Pulmonary complications are observed in about 40% of patients with HES.<sup>2</sup> Approximately 50% of these subjects will have pleural effusions, either from congestive heart failure or thromboembolic disease. About one-third will have interstitial infiltrates in a non-lobar distribution.

Neurologic complications may be seen in about 30% of patients.<sup>2</sup> Two-thirds of these will have diffuse signs of central nervous system involvement, including coma, confusion, delusions, blurred vision, psychosis, ataxia and slurred speech. As many as 20% of these patients may develop hemiparesis, presumably from emboli released from thrombi overlying damaged endocardium. Another 20% of patients with neurologic involvement will develop a peripheral neuritis.

Cutaneous lesions associated with pruritus are found in about 27% of patients.<sup>2</sup> HES associated with urticarial lesions, an elevated level of IgE, and lung disease appears to represent a subset of this syndrome that is particularly responsive to steroids.

Renal lesions are uncommon, but may be seen in about 20% of patients and include pyuria, hematuria, proteinuria and cylindruria.<sup>2</sup> BUN values above 30 may occasionally be seen in the absence of congestive heart failure.

Hepatomegaly can be detected in 85% of patients with hypereosinophilic syndrome, usually related to congestive heart failure.<sup>2</sup> Only about 14% of these patients will have elevations of liver function tests and many of these patients will have infiltration of their liver with eosinophils.

## Treatment

Treatment of the syndrome involves reduction of the eosinophil concentration and aggressive management of organ system complications. Eosinophilia alone requires no treatment, though such patients should be evaluated at 3-6 month intervals for organ system dysfunction.<sup>32</sup> Oral prednisone (1 mg/kg/day) for 1-2 weeks followed by alternate day prednisone at the same dose for three months has been suggested as initial therapy. Improvement indicates that the prednisone should be continued in a gradually tapering dosage schedule. Progression of organ system complications demands more aggressive chemotherapy, usually with hydroxyurea,<sup>32,33</sup> but cyclophosphamide, vincristine, 6-mercaptopurine and busulfan have also been em-

ployed.<sup>29,32,33</sup> Antithrombosis therapy with dipyridamol or aspirin, anticoagulant therapy with coumadin, and plasmapheresis have also been utilized though without apparent benefit.<sup>29</sup> Patients with heart failure may require diuretics and inotropic agents. Left ventricular dysfunction associated with valvular incompetence, even when severe, may respond to surgical management of the defective valve.<sup>31,34</sup> In the National Institutes of Health series discussed above,<sup>1</sup> survival increased from 20% at five years in a group of historical controls to 80% at five years in the patient population that had received aggressive medical and surgical therapies.

## Summary

Hypereosinophilic syndrome occurs when prolonged eosinophilia causes organ system complications. These complications appear not to be related to the magnitude of eosinophilia, but may relate to the magnitude of tissue invasion and degranulation of eosinophils. As a corollary, pathologic sequelae may occur when excessive amounts of certain mediators of eosinophils are secreted into tissues, such as major basic protein, eosinophil cationic protein, and peroxidase. Although almost any organ system can be involved, cardiac and central nervous system complications are associated with more severe disease, whereas an HES subgroup associated with urticaria, pulmonary lesions and elevated levels of IgE is usually responsive to treatment. Initial treatment consists of steroids and standard medical care for any organ system complications. When further therapy is required, hydroxyurea and other cytotoxic drugs along with appropriate surgical interventions may be employed. Aggressive medical and surgical management of hypereosinophilic syndrome has apparently increased survival at five years from 20% to 80%.

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## INFECTIOUS MONONUCLEOSIS cont'd from page 349

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## VIRGINIA AUTHORS

### Urinary Complications of Cyclophosphamide Therapy: Etiology, Prevention and Management. Frederick A. Klein, MD, and M. J. Vernon Smith, MD, *Richmond*.

Cyclophosphamide is a well established cytotoxic drug used in the treatment of lymphoproliferative disorders, certain solid tumors, and non-neoplastic disorders such as the nephrotic syndrome, systemic lupus erythematosus, and rheumatoid arthritis. Hemorrhagic cystitis can be a complication of this drug in from 2% to 40% of patients so treated. At times, the hemorrhage may be severe, protracted, and life-threatening. Cyclophosphamide therapy has also been implicated as the causative agent in 32 cases of carcinoma of the bladder and three cases of carcinoma of the renal pelvis. *South Med J* 1983; 76:1413-1415

### Hyperfunctioning Cystic Parathyroid Glands: CT and Sonographic Findings. Adrian G. Krudy, MD, John L. Doppman, MD, Thomas H. Shawker, MD, Allen M. Spiegel, MD, Stephen J. Marx, MD, and Jeffrey Norton, MD, *Bethesda, Maryland*; Marcus Schaaf, MD, *Washington, DC*; Morton L. Moss, MD, and Michael A. Weiss, MD, *Fairfax*; and Stephen H. Schachner, MD, *Falls Church*.

Four functioning cystic parathyroid glands were evaluated with computed tomography (CT) and sonography in four patients, only one of whom had prior surgery. Sonography demonstrated solid lesions of decreased echogenicity with fluid-filled cavities near the lower thyroid poles or in the posterosuperior mediastinum. On CT the cystic parts of the lesions were of low attenuation (1-44 H), often with a well defined wall that was better demonstrated after intravenous contrast administration. Fine-needle aspiration biopsy of two of the cystic parathyroids revealed elevated parathyroid hormone levels. These lesions probably represent degenerating adenomas rather than true parathyroid cysts. While the CT and sonographic findings are non-specific, the diagnosis of a cystic parathyroid should be entertained when a fluid-filled lesion is encountered in the neck of a patient with or without hypercalcemia. The diagnosis may be confirmed by assay of parathyroid hormone from the fluid aspirate. *AJR* 1984; 142:175-178

# VIRGINIA MEDICAL

## EDITORIAL

### Wrong Perspective

**W**ITH many great issues begging to be addressed and solved, we squander so much of society's resources in debating, demonstrating, and ranting about the death penalty that we have little left for the main issue—certainty of punishment.

While we anguish over the degree of punishment, we virtually ignore the fact that only 20% of crimes lead to arrest and of those arrests only 10% lead to conviction. Even those few convictions lead to appeals, with the perpetrator of the crime free on bail to perpetrate more crimes so as to pay for both his life style and legal fees.

We have 508,000 inmates in our over-crowded prisons, which often seem to be run more by the prisoners than by the guards—homosexual rape, prison gang wars, 8,000 prisoner escapes per year, prisoner insurrections, etc. We want more stringent prison sentences but then give early parole because our prisons are over-crowded. And then we bridle over funding new prisons and bicker endlessly about where to construct them. "Oh no, not near my town!" is the refrain.

We want better law enforcement, but resent law officers enforcing laws which we ourselves transgress. We vilify "the cops," underpaying and often under-training them, often casting them as dolts in movies and on television but expecting them to come immediately to our aid, to protect us in the direst of circumstances, and to lay down their lives when necessary in our defense.

We say that we want swift and certain justice, but, as the most litigious society in the world, we over-burden the judicial system with thousands of absurd lawsuits. Keeping attorneys employed, we resist more logical and efficient ways of settling relatively minor civil disputes. When one branch of the federal government files suit against another, may not my left hand soon sue my right?

When finally a convicted killer has exhausted innumerable appeals over many years, when the victim of the crime has long since been forgotten by the public, when the family of the victim would long since wish to have mended their emotions, we have debates and shouting matches about the death sentence. Nocturnal vigils against the death sentence are shown on television, as are ugly demands for giving the killer his due. With all of this media hype, society grows more callous.

To rationalize the situation we should debate how to make our police system more efficient against the criminal; how to make our legal and judicial systems function quickly and efficiently; how to allow appeals yet limit them; how to take the repeat criminal off the streets while this is going on; how to fund and where to put our new prisons; and finally and most importantly, how to make the potential criminal realize that any and all crimes—by rich or by poor—lead to swift, sure and fair punishment. This is the agenda to replace the television shows that make folk heroes of the condemned wretches



and present 30-second debates about capital punishment and how it should be inflicted.

The public wheel demands that we debate, decide and act upon the real issues on a priority and not an emotional basis, without side issues and without side shows, so that certainty of punishment will

deter the criminal and console the victim. In this election year both political parties need to receive this message, loudly and clearly, from each of us. Your local political party mass meeting is the place to start.

HENRY S. CAMPBELL, MD

## Waiting for the Other Shoe to Drop

**T**HERE HAS BEEN a hush all over Southwest Virginia ever since the standards of physicians in the remainder of the Commonwealth of Virginia changed to allow the corporate ownership of physicians in the form of HMO/IPAs. The hush is like that of waiting for the other shoe to drop.

In this issue of VIRGINIA MEDICAL is a brochure warning us to watch the contracts of the alternative delivery systems springing up everywhere, to read the contracts carefully and know what we are signing. Other medical organizations are warning us, too. The concept of HMOs and PPOs and IPAs is never questioned; the emphasis is on modifying them to our situation.

In Southwest Virginia there is little or nothing to indicate that we need HMOs or any of the other of these plans. We practice good personal medicine and surgery, and we take good care of our patients. We do this at reasonable cost; we maintain the respect and friendship of our patients. Perhaps because this is the way it is in Southwest Virginia, we are not into contract medicine and a delivery system which is foreign to our beliefs, our goals, and our very reason for going into the practice of medicine in the first place.

In contrast, we see in the remainder of the state a frenzied development of physician groups which are dedicated to the annihilation of one another. This development is such a divisive force that the future of medicine as we have known it is at a horrible risk of being totally undermined. It appears that there soon will be no place for a local medical society because of the severe alienation of mutual respect, a respect that is so necessary for the progress of our profession. The weaknesses created by these divisive forces are invitations for take-overs not only by business and insurance operators but also by the Social Security Administration in a recognizable form of socialized medicine.

The change in the state laws that now legalize contract medicine appears to be a signal for some physicians to engage in commercial aggressiveness

which is fostered more by greed than interest in patient welfare.

When the physicians in the western part of Virginia do reject the invasion of HMOs, there will be no more hush; we will have seen the other shoe for what it is: An attempt to put physicians under the heel of an authority they no longer control.

WILLIAM W. S. BUTLER III, MD

1234 Franklin Road, SW  
Roanoke VA 24016

## The Journal's Role

**A**T a recent meeting of the Editorial Board of VIRGINIA MEDICAL a question arose: what is the goal of VIRGINIA MEDICAL? Should the journal be a vehicle primarily for the publication of scientific medical articles? Or a promotional organ for The Medical Society of Virginia? Should the space be devoted to the work of The Medical Society of Virginia? Should it be a tool for medical politics? How about a journal for the dispensing of general information as to the activities, both individual and collective, of the medical profession in Virginia?

We try to provide a forum for the publication of scientific articles, primarily by and for the physicians in Virginia. But this is only a part of the story. A considerable portion of the space in the journal is dedicated to the news and activities of medicine in Virginia, an area so beautifully orchestrated by our Executive Editor, Mrs. Gray. This includes medical politics and state and national politics that affect medicine and overlaps into the third area, i.e., the work of The Medical Society of Virginia.

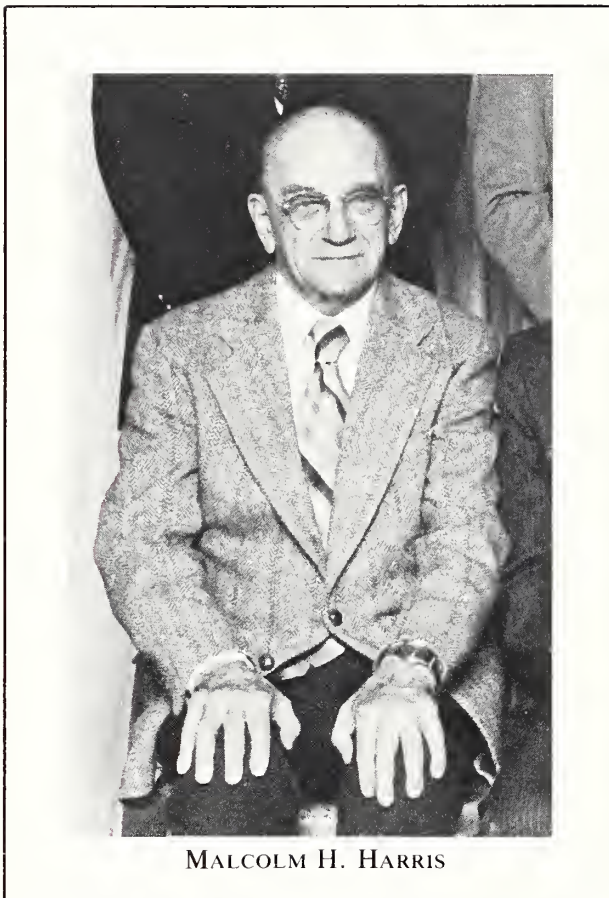
The goal of VIRGINIA MEDICAL is a blend, consisting of relatively equal parts of scientific articles, Medical Society of Virginia activities and general medical news. We hope you like it this way.

EDWIN L. KENDIG, JR., MD

# VIRGINIA MEDICAL OBITUARY

## Malcolm H. Harris, MD

Dr. Malcolm H. Harris, general practitioner in West Point, Virginia, from 1923 onwards, died April 17 in a Richmond hospital after a short illness. He was buried in Louisa County, where he was born 87 years ago. A history of Louisa County from his hand survives him, as does his two-volume account of old New Kent County and the history he wrote of



West Point's First Baptist Church, where his funeral was held.

Educated entirely in Virginia, Dr. Harris was a graduate of the University of Richmond and of the Medical College of Virginia, Class of 1922. During the early 1940s he was West Point's mayor, and he had served on King William County's school and public welfare boards. He was a founder and for many years a board member of Patrick Henry Hospital in Newport News and was a long-time

board member also of the Virginia Baptist Home in Culpeper. For the First Baptist Church in West Point he was faithful member, Sunday school teacher, trustee and deacon.

Dr. Harris was the last surviving founder of the Mid-Tidewater Medical Society. At its first meeting, in 1927, he was named secretary and toiled loyally in that office for 50 years thereafter. Twice the Mid-Tidewater Society physicians also elected him president, in 1948 and 1979, and he had been president of the Tri-State Medical Association and the Virginia Academy of Family Physicians.

A member of The Medical Society of Virginia for 60 years, Dr. Harris had long held membership also in the American Medical Association and the American Academy of Family Physicians.

## Norbourne B. Jeter, MD

Dr. Norbourne Berkley Jeter, past president of the Alleghany-Bath County Medical Society and longtime general practitioner in Alleghany County, died April 14 at his home. He was 82 years old and had retired from practice.

A native of Covington, Virginia, Dr. Jeter was graduated from the University of Richmond and from the Medical College of Virginia, Class of 1928. Except for three-and-a-half years with the Army Medical Corps during World War II, he practiced in the Covington area until his retirement in 1975. He also served for 30 years as plant physician for nearby Industrial Rayon Corporation and Hercules, Inc.

Dr. Jeter's membership in The Medical Society of Virginia spanned more than 50 years, and he was also a member of the American Academy of Family Physicians.

## John Kilday, MD

Dr. John Kilday, for 30 years general practitioner in Alexandria, died April 18 at Circle Terrace Hospital, Alexandria. He was 83 years old and had retired from practice.

A native of Altoona, Pennsylvania, Dr. Kilday was graduated from the University of Pennsylvania and in 1931 earned his medical degree at Temple University School of Medicine, Philadelphia. He



remained in Philadelphia for training at St. Agnes Hospital, afterwards practicing in Altoona and later in Frostberg, Maryland. He then served for three years in the Army Medical Corps during World War II, his tour ending in 1944. Later that year he settled in Alexandria, and in early 1945 began his practice there.

Dr. Kilday came to membership in The Medical Society of Virginia in 1948, through the Alexandria Medical Society. He was also a member of the American Medical Association.

## Memoir of Reuben Simms 1902-1983

*By F. Elliott Oglesby, Sr., MD*

Reuben F. Simms died suddenly from a massive myocardial infarction on April 26, 1983. He was born in West Point, Virginia, on December 8, 1902. He attended William and Mary College through the academic year 1925 and was graduated from the Medical College of Virginia in 1928. He interned at Stuart Circle Hospital and then entered the practice of general medicine. On the day of his death, he had office hours that morning and made a home call that afternoon, as was his daily custom. He did obstetrics until about ten years previous to his death, and he estimated that he had delivered about 2,500 babies.

The words to describe him are good, compassionate and dedicated; these characteristics not only were reflected in his personal and family life but also in the manner in which he practiced medicine. He was not only versed in the techniques of the practice of medicine but also in its art, a gift not shared by all the profession.

Other than his family and his practice, his devotion to duty was his involvement in the Lion's Club. He had a perfect attendance for his 43 years of membership, was vice president and then president of his local club, and elected to the high office of district governor. He was a charter member of the Virginia Academy of Family Physicians organized in 1947 and maintained his membership and activity in it throughout his lifetime. He was a member of Emmanuel Episcopal Church.

No closer friend and confidant have I had in my lifetime. I had the privilege of being a student of his in the old Dermatology and Syphilology Clinic at the Medical College of Virginia and of being his primary care physician during his latter years. Both were learning experiences, which I cherish. Life goes on,

but there are always those who can never be replaced: the one-of-a-kind persons. Such was Reuben F. Simms. No one can ever possibly know the amount of good his life generated. It is trite to say that he will be sorely missed, but he will.

## Memoir of A. S. Thompson, Jr. 1933-1983

*By Edwin L. Kendig, Jr., MD, and  
Carolyn M. McCue, MD*

Albert Sidney Thompson, Jr., MD, a beloved colleague, died on June 17, 1983, the victim of an automobile accident sustained while en route to spend Father's Day in his parents' home.

Dr. Thompson was born in Bluefield, West Virginia, received his degree in pharmacy from the Medical College of Virginia in 1954 and was graduated in medicine from the same institution in 1960. He served in the United States Armed Forces, first as ensign and later as second lieutenant, with assignment to the United States Public Health Service.

Dr. Thompson received his pediatric training at the Medical College of Virginia hospital, serving with distinction as chief resident during his fourth year.

For the past 19 years, he had been engaged in the practice of pediatrics in Northern Chesterfield county, with a large and devoted following. During this period he served a term as president of the Richmond Pediatric Society and was a member of several important committees of The Medical Society of Virginia.

Dr. Thompson was also active in the affairs of the Richmond Academy of Medicine and under the auspices of this organization was instrumental in developing the manual, "Pre-Hospital Treatment Protocols", currently in use by Richmond area emergency technicians.

His community activities included service as a ruling elder of the Crestwood Presbyterian Church, of which he was also a charter member. He was also chairman of the board of trustees of St. Michael's School.

Dr. Thompson's passing will be a great loss to the medical profession and to the community as a whole.

He is survived by his wife, Mrs. Nancy Grubb Thompson, and a son, Albert Sidney Thompson III.

# WHO'S WHO

**Dr. Edward W. Hook**, Charlottesville, is the new president elect of the American College of Physicians and has also been named a member of the United States delegation to the US-Japan Cooperative Medical Science Committee. The election by his ACP peers occurred at the college's annual scientific meeting in Atlanta in April. His appointment to the delegation was made by the Secretary of State.

Dr. Hook is chairman of the Department of Internal Medicine at the University of Virginia. A specialist in infectious diseases, he is widely known for his research in salmonella and other intestinal infections. He has recently been in the spotlight as one of the ten physicians who were convened by the Society for the Right to Die to develop guidelines for physicians in dealing with terminally ill patients and whose report received voluminous media attention.

**Dr. Paul G. Rochmis**, Fairfax, has been elected president of the Medical Council of the Washington Metropolitan Area, an organization of the six medical societies of Alexandria and the District of Columbia and the counties of Arlington, Fairfax, Montgomery and Prince George. Dr. Rochmis also is president for 1984 of the Medical Council of Northern Virginia and was president in 1983 of the Fairfax County Medical Society. He is chief of rheumatology at the Fairfax Hospital.

To **Dr. Belle DeCormis Fears**, Accomac, went the 1984 Outstanding Citizen Award of the Eastern

Shore Chamber of Commerce for her "unselfish service to the Eastern Shore of Virginia community." Prior to her recent retirement, Dr. Fears was for 18 years director of the Accomack and Northampton Health Departments.

New president of the Virginia Society of Internal Medicine is **Dr. William C. Branscome**, Staunton. Serving with him are **Dr. John W. Knarr**, Pulaski, president elect, and **Dr. W. Wayne Key, Jr.**, Richmond, vice president.

**Dr. Carrington Williams, Jr.**, is serving as president of the Northern Neck Medical Association for 1984, and **Dr. Robert W. Poole**, who in *VIRGINIA MEDICAL*'s March issue was erroneously listed as the association's new president, is its president elect. Serving with them are **Dr. David H. Summers**, vice president, and **Dr. Eugene J. Wolski**, secretary-treasurer. Dr. Wolski practices in Callao, the others in Kilmarnock.

After 36 years of surgical practice, **Dr. David B. Corcoran** of Suffolk retired from the operating room regimen early this year, opting for travel, reading, golf and sailing. He started his new life of relaxation with a three-week visit with friends in Florida. This month he and his wife will do some European sightseeing. In future he will likely be travelling to New Jersey, where a son now lives, and to Sun Valley, Idaho, the home of a daughter. (Another son elected to stay in Suffolk.) And there will be frequent excursions to the Corcorans' beach-side house in Sandbridge.

Born in Central Islip, Long Island, New York, Dr. Corcoran was educated at Fordham and took his doctorate in medicine at the State University of New York, Class of '40. His residency at St. Vincent's Hospital in New York was interrupted by service in the European Theater during World War II, from which he emerged Major Corcoran USAMC. To complete his surgical training, he went to the McGuire Veterans Administration Center in Richmond. Hearing of an opening at the Lakeview Clinic in Suffolk, he applied for the job, got it, and the residents of Suffolk have benefitted from his care ever since.

**Dr. Benjamin F. Allen, Jr.**, Culpeper, has been elected to fellowship in the American Academy of Orthopaedic Surgeons.

**Dr. Edgar N. Weaver**, Roanoke neurosurgeon, is one of six persons named to a committee to search for a new president for the University of Virginia to succeed Frank L. Hereford, Jr., who is to retire in August 1985.

New president of the Hanover County Medical Society is **Dr. Jethro H. Piland, Jr.**, Mechanicsville.

**Dr. W. R. Chitwood**, Wytheville, is the coauthor of a recently published pictorial history titled *An Album of Wythe County, Virginia*. The book was inspired by the photographic collection of the late F. B. Kegley, Wythe County lawyer, historian and photographer; Mrs. Kegley collaborated with Dr. Chitwood on the book, which drew also on photographs donated by



citizens of the area. Included in the book are 144 black and white photographs of Wythe County buildings. It was published by a Missoula, Montana, firm specializing in local pictorial histories.

"It's been 40 years since I graduated from medical school and I'm just tired of working," explained **Dr. William A. Sadler** of Mathews to a reporter from the *Gloucester-Mathews Gazette-Journal* who was inquiring about Dr. Sadler's retirement at the close of 1983. The leavetaking was not altogether easy, however, the physician admitted. "I've taken care of generations of people," he reflected, "and I do get to feeling a little sentimental."

Dr. Sadler was graduated from the Medical College of Virginia in 1944, then trained at Elizabeth Buxton Hospital in Newport News. There he met his wife, Lynn Powell, RN, who was the hospital's supervisor of obstetric nurses. Mrs. Sadler helped her husband set up his practice in Mathews Court House, was his partner in it thereafter, and retired with him. They have three children, all grown.

**Dr. G. Edward Chappell, Jr.**, Harrisonburg, has been elected to fellowship in the American College of Surgeons.

After 35 years in the field of preventive medicine and public health, **Dr. George Moore** of White Stone retired early this year from his post as director of the Northern Neck District Health Department.

The Arlington County Medical Society has elected this new slate of officers: **Dr. Robert G. Bullock**, president; **Dr. Roger D. Cornell**, president elect; **Dr. Frank A. Petrone**, vice president; **Dr. William L. Stone III**, secretary; and **Dr. Catherine S. Casey**, treasurer.



Time to relax with friend Gretchen: Dr. Corcoran  
Photo by Linda McNatt for the *Suffolk News-Herald*



After 40 years, a team retires: Dr. and Mrs. Sadler.  
Photo courtesy the *Gloucester-Mathews Gazette-Journal*

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## From the President: Why Organized Medicine?

**R**ECENTLY I was in Taiwan on a business trip involving one of the medical schools in that country. Prior to going, I had asked if I could visit with the equivalent of a county, state or national medical association.

While there, I learned that none of the above organizations existed in such a way that it would be analogous to one of our organizations. They did indeed have a national scientific organization, but nothing socioeconomic or political in nature.

During my absence from the country, the AMA had mounted a tremendous lobbying effort against mandatory assignment of benefits for Medicare patients. Ultimately, this proved to be successful, as you know by now.

An issue which is of great concern to many physicians is that of the "Physician Attest" which is the certification requirement now placed on the front of all Medicare patient charts. The full ramifications of this have not yet been determined, but there is concern that it may greatly increase physician liability without serving any useful purpose. The AMA is working with various government officials to modify/improve the wording.

Since returning from Taiwan, I have thought of the "Health Manpower Agenda" for the American people being developed by the AMA at the cost of several millions of dollars and many man hours. People from well over 100 various organizations, from religious to business, union, medical and others, have participated. This will be a health plan for the next 20 years.

Who on the JCAH worked to preserve the terminology of the medical staff in regard to membership? Those members of the JCAH from the AMA led the charge.



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# VIRGINIA MEDICAL

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Who attempts to look after the interests of all doctors, not just those in organized medicine? The AMA, yes, but also your state and local medical associations.

Some of the cynics feel that these organizations are primarily interested in protecting the financial interests of doctors. Certainly that is one thing they do but far from the most important.

These recent developments have made me realize even more how fortunate we are to have this triumverate of medical organizations to look out for doctors and patients. Taiwan has none of these. They do have a closed hospital staff situation for all hospitals, both private and public. Those without privileges cannot admit patients to hospitals and generally do not participate in continuing education within the hospitals. As a result, there is little concern about the quality of care by physicians outside or inside the hospitals, especially the private hospitals. There is no health care agenda for the Taiwanese people, there is no JCAH to be concerned about standards within hospitals, no AMA Council on Scientific Affairs to evaluate drugs or therapeutic modalities, no Council on Graduate Medical Education, etc.

As a result, I have become an even stronger advocate of organized medicine at all levels. I believe that all the innovative, worthwhile efforts of the AMA are more important than ever for our profession and our patients.

Therefore, I urge all of you to jump in, become an active member of the medical team, and make your influence felt.

**C. BARRIE COOK, MD, PRESIDENT,  
The Medical Society of Virginia**

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# House Call

The Medical Society of Virginia's leadership goes to Washington to give its annual Congressional Luncheon.



Senator Paul S. Tribble cozies up to Dr. F. Ashton Carmines.





As tourists stream past, four Medical Society of Virginia councilors pause on the balcony of a Capitol rotunda: from left, Dr. John A. Owen, Jr., and Dr. William W. Regan, both new to Council this year, and two veteran councilors, Dr. Nicholas G. Colletti and Dr. J. Thomas Hulvey.

**T**he photographs on these four pages fall far short of complete coverage of The Medical Society of Virginia leadership's visit to Capitol Hill. Many more physicians made the trip than are shown here—22 of them altogether, and all but one of Virginia's legislators showed up. The absentee was Rep. James R. Olin (R-6th district), whose aide stopped by to report he was testifying at a hearing.

The two senators, John W. Warner and Paul S. Trible, sat at the head table with President C. Barrie Cook, while the congress-

men were seated about the Mike Mansfield Room in the company of MSV councilors from their districts.

In the space of one hour exact, lunch was served, Dr. Cook welcomed the guests, and each of the eleven legislators spoke. Their remarks rang with an extra geniality in this election year, particularly the expressions of appreciation for physician support.

Last to speak was Rep. Frank R. Wolf (R-10th district), who departed from political issues to reveal that at his instigation, a component medical society in Upper Virginia was exploring the possibility of

In a Capitol hallway, Ben Franklin looks benignly on three Medical Society of Virginia presidents, past and present: from left, Dr. Raymond S. Brown, Dr. W. Leonard Weyl, and Dr. C. Barrie Cook.







At left, in pre-lunch palaver, Dr. Michael A. Puzak (half-hidden), Dr. Richard L. Fields, Dr. Russell D. Evett, and Dr. Leon I. Block.

Below left, Dr. William S. Burton listens to Rep. H. H. Bateman (R-1st district). Directly below, Congressman Dan Daniel (D-5th district) finds an attentive audience in Dr. William J. Hagood, Jr., (center) and Dr. Anthony J. Munoz.



"adopting" a needy hospital in El Salvador.

In an official visit to the Central American country, Wolf related, he was everywhere dismayed by the country's poverty, but it was the critical needs of a hospital at Santa Ana that struck him most forcibly. To this hospital are brought the wounded from heavy fighting in the area, Wolf explained, but the staff had long since

run out of medical supplies with which to treat them.

In a recent appearance before the Arlington County Medical Society, he described the hospital's plight, with the result that Dr. W. Leonard Weyl is heading up an effort to help, and three Arlington County Society members were designated to journey to Santa Ana to evaluate the hospital's needs—Dr. Catherine S. Casey, Dr. Philip Bor-

ges and Dr. Javier A. Vasquez. Wolf assigned his aide, Thomas M. Fulcher, Jr., as the delegation's fourth member. With them went \$5,000 worth of sutures, dressings, sponges, and other such supplies.

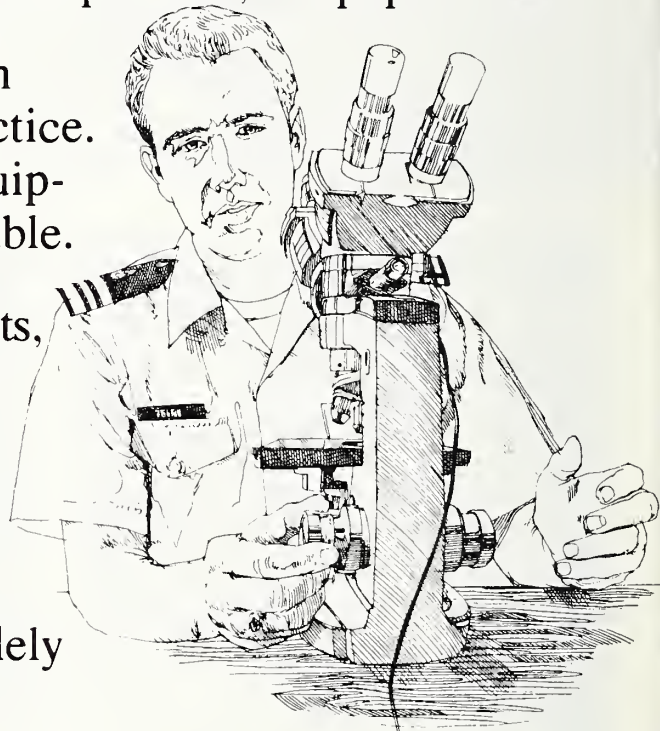
El Salvador's other hospitals are in similar straits, Wolf concluded, and he urged the physicians listening to encourage their local societies to emulate the Arlington County Society's project. A.G.

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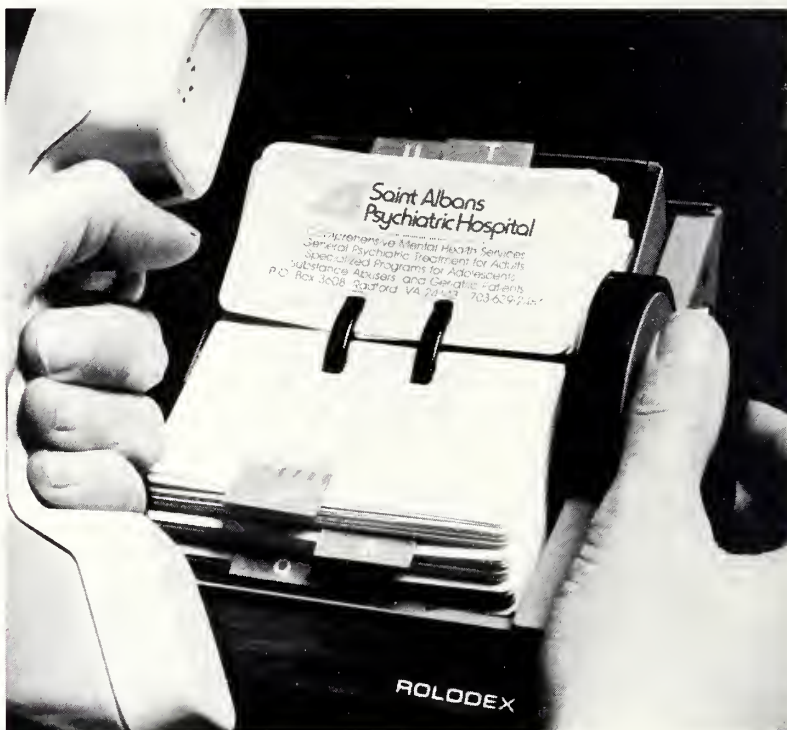
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# Management of Obesity

James N. Cooper, MD,  
and Ilene R. Robeck, MD  
*Falls Church, Virginia*

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Successful treatment of obesity depends on a patient who is strongly motivated to fight this recalcitrant disease by undertaking a long-term program of diet, behavior modification, and exercise. The authors review the etiology of obesity and describe current therapies.

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**O**BESITY is a common problem in Virginia and throughout the United States and represents one of the most difficult diseases to treat. The Nutrition Committee of the Virginia Council on Health and Medical Care has developed a brochure giving guidelines for choosing a weight-loss program. (See page 386.)

Obesity has traditionally been defined with reference to actuarial height and weight tables supplied by insurance companies, which reflect the weight consistent with a normal life expectancy. It is arbitrarily defined as individuals who are 20% or more above their ideal body weight. The role of edema and fluid retention, however, must always be assessed in the patient who is overweight. In addition, there is a small minority of overweight patients whose increased weight is due to increased

From the Department of Medicine, the Fairfax Hospital, Falls Church, Virginia, and Georgetown University Medical Center, Washington, DC. Address correspondence to Dr. Cooper at 3300 Gallows Road, Falls Church VA 22046.



Photo by Robert Stewart



muscle mass, i.e., athletes. Another measurement used to assess body fat is the body mass index, which is equal to weight/height<sup>2</sup>. Using this measure, obesity is defined as a body mass index greater than 27 kg/m<sup>2</sup> in males and 25 kg/m<sup>2</sup> in women.

It is estimated that approximately 5%-25% of adult males and 10%-55% of adult females are obese. Approximately 2%-15% of children are obese. The incidence of obesity is increased in women of low socioeconomic status and women who are recent immigrants to the United States, regardless of ethnic background and socioeconomic status.<sup>1</sup>

While controversy surrounds the exact statistics with reference to morbidity and mortality associated with obesity, there appears to be a clear-cut increase in the risk for cardiovascular disease, liver and biliary tract disease, and maturity-onset diabetes in patients who are at least 20% above ideal body weight. Other medical problems that have been exacerbated by obesity include congestive heart failure, hernia, hypertension, hyperlipidemia, thrombophlebitis, arthritis, pulmonary disease, toxemia, and infertility.<sup>2,3</sup>

There has been a great deal of research and interest recently in the relationship between obesity and hypertension. The mechanism of this association is unclear. Some hypotheses include 1) the hyperinsulinemia associated with obesity promotes sodium retention by the kidneys; 2) there is increased sympathetic tone and turnover of epinephrine associated with over-feeding; and 3) the lack of physical activity so prevalent among the obese may exacerbate the hyperinsulinism and sodium retention found in obese patients. Many studies have demonstrated improvement in blood pressure control when weight loss in obese patients with hypertension occurs, especially when a program of exercise and sodium restriction is simultaneously adhered to.<sup>4-8</sup>

### Etiology

Obesity is a disease that is multifactorial in origin. Heredity, food availability, psychologic stresses, familial eating patterns, and societal pressures all play a role in its development. Obese patients can be separated, in general, into two broad categories that carry with them different etiologic and prognostic implications.

Hyperplastic obesity is characterized by a disease in which patients present as adults at greater than 50% of their ideal body weight and have a history of lifelong weight problems. There is an

increase in cell number as well as cell size, and the fat distribution is peripheral as well as central. These patients have a poor long-term response to treatment, with only 5% maintaining their weight loss for three or more years.<sup>9,10</sup>

A number of studies have explored possible etiologic factors in this subset of obese patients. Pettitt et al studied excessive obesity in the offspring of Pima Indian women with diabetes during pregnancy. They found that children of women who had diabetes during pregnancy had a higher prevalence of obesity than children of pre-diabetics and non-diabetics. In fact, at 15 to 19 years of age, 58% of the offspring of diabetics weighed 40% or more of their desirable weight as compared with 17% of the offspring of non-diabetics and 25% of the offspring of pre-diabetics.<sup>10</sup>

In another study, Charney et al linked obesity with weight during the first six months of life, finding a strong correlation between infant weight and adult weight.<sup>11</sup> This may be influenced by food availability, as demonstrated in the studies done on infants born during the Dutch famine in 1944-1945. This study showed a decreased incidence of obesity, while infants born during the first summer of increased food availability showed an increased incidence of obesity.<sup>10</sup>

Obesity that begins during adulthood is termed hypertrophic and is characterized only by an increase in cell size.<sup>12</sup> It is generally moderately severe, and fat distribution is central. Prognosis is fair, with variable rates of response in terms of long-term weight reduction that are higher than those seen with hyperplastic obesity.

### Treatment

When faced with an obese patient in practice, the initial evaluation should be directed toward uncovering the existence of contributory medical problems, such as hypothyroidism, Cushing's disease, Stein-Leventhal syndrome, Klinefelter's syndrome, and drug ingestion. (Drugs such as phenothiazines, tricyclic antidepressants, and cypheptadine have been associated with weight gain.)

Once associated metabolic and pharmacologic factors have been ruled out, one should focus on the following areas: 1. evaluation of the patient's motivation; 2. determination of the duration of the patient's obesity; 3. previous responses to attempts at weight loss. Poorly motivated patients, patients with childhood-onset obesity, and patients with poor track records with previous diets carry the worst prognoses.

In all patients, a program consisting of diet,

behavior modification, and exercise is the cornerstone of therapy. A dietitian should be included in the health team. If the patient is less than 100 pounds overweight, a well-balanced, calorically restricted diet should be tried initially. The patient can follow the diet by counting calories or the use of food exchanges. In general, after the initial diuresis that occurs at the initiation of dieting, a 1200-calorie-per-day diet for men and a 1000-calorie-per-day diet for women will result in a 1-to-2-pound weight loss per week.

There are many behavior modification programs for weight loss. Some concepts common to all of these programs:

- Keep a daily log of both food intake and exercise. The log for food intake should consist of columns for time and duration of eating; food quantity; number of calories; where the food was eaten; how the individual felt before, during and after eating.
- Avoid going into tempting places, such as bakeries, especially early on in the diet.
- Always eat sitting down.
- Eat slowly, chewing each bite before taking the next, and place utensils down between bites.

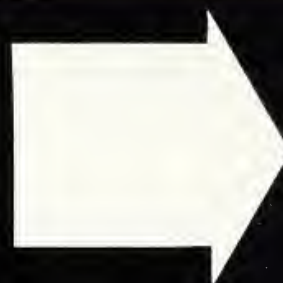
A minimum of 20 minutes exercise three times a week should be encouraged. Many patients are better able to follow through with this when formally enrolled in an exercise or sports program.

Many patients will be able to take weight off and keep it off more successfully by participating in one of the many self-help groups set up in most metropolitan and suburban areas.<sup>13,14</sup>

Drug therapy should be discouraged. Although there are many weight-loss drug products on the market, there is no evidence in the literature that they will help promote a sustained weight loss. Their positive effects, when present, are transient.

For the patient with morbid obesity, more extreme measures may be necessary, especially if there are medical problems caused by the obesity that require immediate treatment. As mentioned previously, these patients frequently have hyperplastic obesity beginning in childhood. Traditional weight-loss techniques have been unsuccessful in this group of patients, who often are the subjects of more radical medical and surgical techniques.

Nutritionally sound, commercially available low-calorie diets have been used with increasing frequency over the past decade. The newer, high-quality protein products are a relatively safe and effective way to initiate weight loss in the morbidly obese individual who has not succeeded with the



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more traditional approaches. They should be distinguished from the "liquid protein" diet popularized in the late 1970s by Linn and Stuart,<sup>15</sup> which consisted of a very poor grade of protein given to patients with varying amounts of medical supervision. Of the patients who died on this diet, all were on it for more than two months and many had a preexisting underlying illness.<sup>16</sup> The causes of death included myocardial protein degradation, electrocardiographic changes characterized by QT prolongation and resultant ventricular tachycardia, and electrolyte disturbances involving calcium, potassium, magnesium, and sodium.<sup>17</sup>

The risk of death with the current generation of very-low-calorie diets is low. There have been some studies showing improved cardiac performance while on these diets. The main disadvantage of these diets remains the low overall, long-term success rate when the patient is unwilling or unable to modify eating behavior and exercise habits formed over a lifetime.<sup>18</sup> Patients receiving these diets require careful medical supervision and weekly laboratory testing under the direction of physicians experienced with very-low-calorie, protein diets. These diets are expensive. Once again, behavior modification and an exercise program should be part of the regimen.

The surgical approach to obesity has consisted of jaw wiring, jejunoileal bypass, and, most recently, gastric stapling. Unfortunately, the positive effects of weight loss following jejunoileal bypass have been outweighed by the high incidence of side effects. These include diarrhea, electrolyte disturbances, folate and B<sub>12</sub> deficiency, nephrolithiasis, cholecystitis, and such hepatic structural abnormalities as cirrhosis. Gastric restricting procedures are now being used for the surgical treatment of obesity. Their place in the treatment of obesity is still being evaluated.<sup>19-22</sup>

Only about 10% of patients have maintained their weight loss at three-year followup. This figure is not meant to discourage physicians from treating obese patients, because for those patients who are able to maintain weight loss, an impressive improvement in quality as well as quantity of life is noted. However, these statistics remain as a cautionary reminder that successful therapy depends on the selection of well-motivated patients willing to participate in all aspects of a weight-loss program.

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# Food Safety: Regulations, Research, and Results

Joseph F. Borzelleca, PhD,  
*Richmond, Virginia*

**A**MERICANS are blessed with an abundance of wholesome, available and affordable foods. We are unique among nations. But Americans are becoming food-conscious and are asking questions. Is this or that food nutritious? Is it good for me? Is it safe?

Safety in a food is the absence of any adverse health effect under defined conditions of use. A food or beverage may not elicit any harmful effect when consumed normally but could cause severe effects if consumed in excessive amounts or if injected. For example, a certain amount of water is necessary for life. We are encouraged to drink eight glasses (2 liters) per day. If 10-20 liters were drunk at a single sitting, water intoxication and death would occur. If the 2 liters were placed in the lungs, death by drowning would follow. Safety should be viewed relative to the exposure conditions.

Have foods been consumed that were subsequently shown to be harmful? Yes. Sassafras tea was used as a beverage and as a tonic for many years, but safrole, a chemical found in sassafras, was reported to cause tumors in rats, so sassafras was removed from the market. Butter yellow was used to color foods for many years; when it was reported to cause tumors in animals, it also was removed. Removal may be initiated by the manufacturer or by the government.

The regulations controlling the safety of food are many and complex; they include the Food, Drug and Cosmetic Act, Meat Inspection Act, Poultry Products Inspection Act, Federal Insecticide, Fungicide and Rodenticide Act, and their amendments. There are three federal agencies involved in en-

forcement: the Food and Drug Administration (FDA), Food Safety and Inspection Service of the Department of Agriculture, and Environmental Protection Agency.

The Federal Food and Drugs Act of 1906 gave the federal government the authority to regulate the safety of our foods. Food was adulterated and declared illegal if it contained any ingredient that was poisonous or injurious to health. This act was replaced by the Federal Food, Drug, and Cosmetic Act of 1938. The Food Additives Amendment of 1958 required premarketing approval of food chemicals clearly identified as food additives. The following categories of food and food ingredients were defined: 1) food additives; 2) color additives; 3) animal food and drug additives; 4) pesticide residues; 5) substances generally recognized as safe by qualified experts (GRAS); 6) substances approved by the FDA or the Department of Agriculture for use in food prior to 1958 ("prior sanction"); and 7) naturally occurring substances, to which was later added environmental contaminants. Substances are permitted either with or without restrictions on use or are banned. In 1969, the FDA banned cyclamates, a GRAS substance, but shortly thereafter began a critical reevaluation of the GRAS list. This review lasted 11 years, and more than 400 substances were reviewed. Some had their GRAS status affirmed; others were assigned food additive status, subjected to appropriate restrictions, or banned. The FDA is contemplating cyclic review of all additives.

The assessment of risk of injury, or safety evaluation, involves the critical review of a large body of data by toxicologists, who are trained for this purpose. They will establish a safe level of exposure for humans and may also determine the risk of a particular effect.

Safety data can be derived from human studies, epidemiological or experimental, or from animal studies. Human studies are preferred since the concern is human health, but ethical, moral and legal constraints prohibit these except under special conditions. Epidemiological studies may suggest a correlation or association between two events, i.e., exposure to a chemical and manifestation of a disorder, but these studies cannot prove a cause and effect relationship, for example, cigarette smoking and lung cancer. Effects observed in animals are extrapolated to man. This is scientifically valid and is based on the "oneness in nature" concept. Most human physiologic processes are very similar, sometimes identical, to those in animals. These include respiration, digestion, circula-

From the Department of Pharmacology/Toxicology, Medical College of Virginia/Virginia Commonwealth University. Address correspondence to the author at Box 613, MCV Station, Richmond VA 23298.

Submitted 1-3-84.



tion, reproduction and biotransformation, (the chemical mechanisms used by the body to break down foreign chemicals). There are also anatomical similarities.

All living forms studied in the laboratory are treated with respect by competent, conscientious and concerned scientists. Lives are *not* wasted. The number of animals used is dictated by sound scientific principles and the nature of the experiment. Husbandry conditions, such as feed, water, lighting, temperature, caging, are carefully controlled for humanitarian and scientific reasons. Abuse is not tolerated. Animal studies are very expensive and time-consuming. Alternatives are being investigated intensively, and eventually, perhaps, some of these may play a significant role in safety assessment.

What are the components of a safety evaluation program or the assessment of the risk of injury of a food chemical? How much data does a toxicologist need before a decision can be made? A typical program will include all or most of the following items.

1) *Chemical and physical characterization*: chemical name, structure, sources, purity, stability, analytical procedures, manufacturing processes, commercial specifications.

2) *Human exposure*: history of use, natural occurrence, quantities produced, patterns of use, anticipated or known intake, intake by population subgroups, for example, young, old, female, male.

3) *Animal tests*: acute (single dose) oral toxicity; repeated dosing (14-28 days); subchronic dosing (90 days—short term feeding); chronic dosing (life-time feeding, usually 24 months); reproduction and teratology (effects on conception, gestation, intrauterine development, parturition, postnatal development); kinetics (disposition of the chemical in the body); genetic toxicology (effects on DNA); special studies when indicated, analysis of data, extrapolation to man.

4) *Effects in humans*: clinical observations when appropriate, epidemiological studies, inadvertant exposure, anecdotal reports.

These studies can take five to ten years to complete and cost more than \$1 million. Most approved food and color additives have been subjected to extensive study. If the food chemical evaluated under appropriate test conditions causes cancer in animals or in man, it cannot be used in foods because the Delaney Amendment to the Food, Drug and Cosmetic Act specifically prohibits it. But what about saccharin? In properly conducted studies in rats, it was reported to cause bladder cancer. Why

was it not banned? The Food and Drug Administration attempted to ban the use of saccharin but Congress intervened, in response to the public, and extended its use. Congress makes the laws and the exceptions. Does this mean that saccharin is a threat to health? What are the benefits and risks for its use? If it causes cancer in rats at very high doses, will it cause cancer in humans at low doses? Very high doses are used in animals in an attempt to define a toxic or adverse effect; one administers as much as the animals can tolerate, without killing them, to elicit a response. Also, high doses are administered because animals don't live as long as humans.

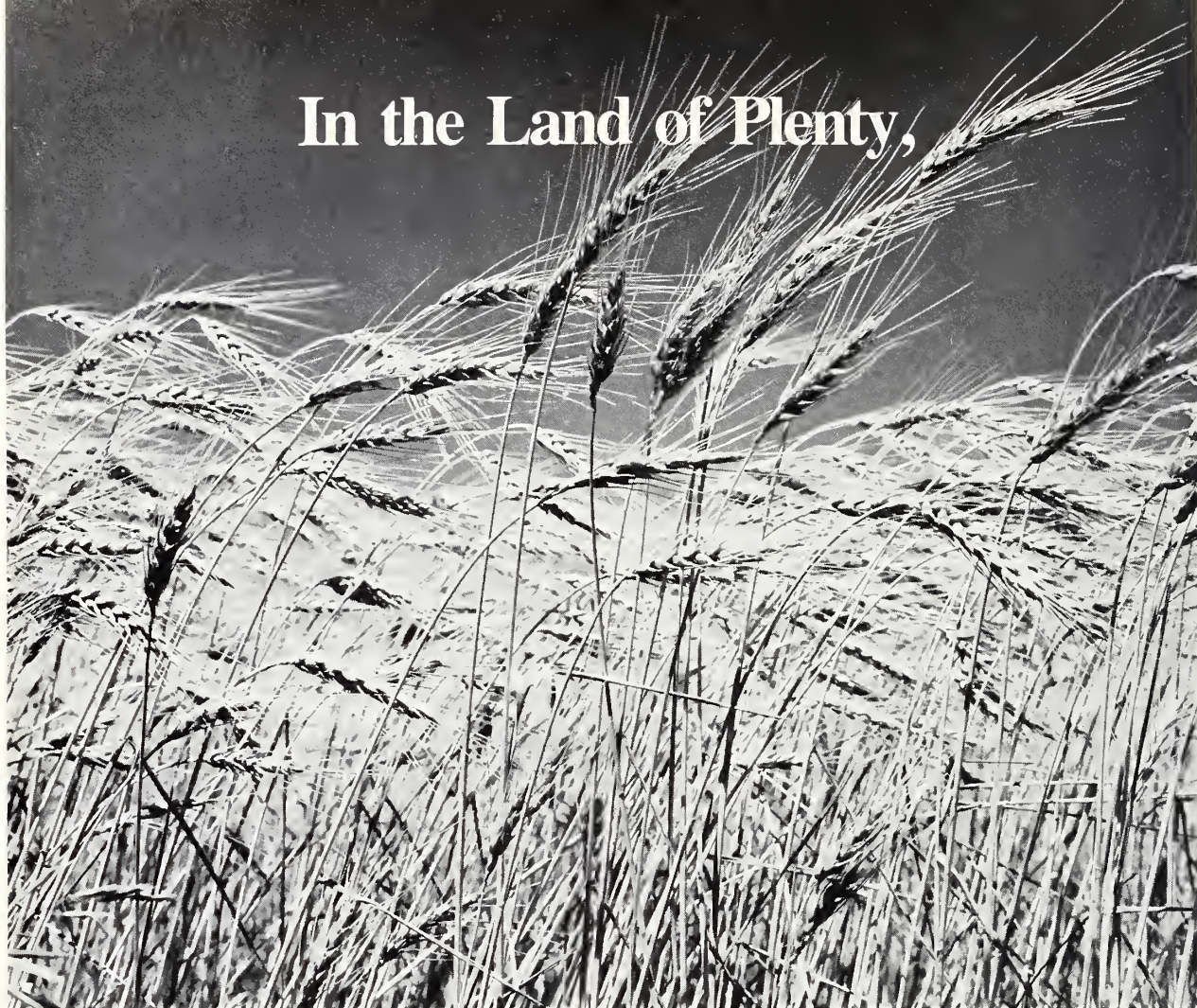
However, high doses of a chemical are often biotransformed in the body in a manner different from smaller doses. It may be scientifically unsound, then, to extrapolate from high-dose animal studies to low-dose human exposure. Not all chemicals that cause cancer in rats or mice cause cancer in humans. Epidemiological data on saccharin users have not revealed a higher incidence of cancer. The risk to human health does not appear to be great, if it exists at all. Do the benefits outweigh the risks? The individual consumer must decide. If the concern is calories, not limitation on intake, then the benefits are obvious: several six-packs of no-calorie or low-calorie drinks vs one can of regular beverage sweetened with sugar. The benefits could be greater for diabetics and others forced to limit intake of sugar.

The critical issue is risk *to whom* and benefit *to whom*. An individual may decide in a case such as saccharin, or society may decide. If the adverse effect produced is not cancer, the toxicologist will identify a safe level of exposure. This is based on the application of a safety factor to the highest dose that did not produce an adverse effect in animals. Another approach is to approximate the risk of an adverse effect at a certain dosage level. This approach is still in the development stage.

Articles on food safety appear almost daily. "Caffeine causes cancer and malformation." "Salt causes high blood pressure". "Sugar causes diabetes". "Butter causes atherosclerosis". What are your patients to believe? Tell them to stop worrying about food safety. Experts in universities, in food companies, in agricultural cooperatives and in government are devoting their talents and their time to this issue. Americans should enjoy in moderation their fruitful bounty.



# In the Land of Plenty,



## Anorexia Nervosa

Julian T. Brantley, MD, Gary Litovitz, MD,  
and Stephen Schachner, MD, *Falls Church, Virginia*

**A**NOREXIA nervosa is a syndrome characterized by volitional self-starvation in young women who express an extreme fear of becoming fat. Because of various unusual features, including associated endocrinologic disturbances, a predominance of higher socioeconomic class cases, the presence of a fairly typical anorexic personality, and a dramatic increase in prevalence over the past

two decades, anorexia nervosa has attracted much attention recently from the medical profession and the general media. Anorexics frequently are preoccupied with attaining the kind of slim, athletic physical condition that is currently in vogue, suggesting a cultural component in its pathogenesis.

The syndrome was first described in 1689 by Richard Morton and considered by him to be a "nervous atrophy from sadness and anxious cares". William Gull later elaborated on and named the disorder. Appreciating that the family played a role in the illness, he recommended that patients be separated from their families during the treatment process.<sup>1</sup>

In 1914, Simmonds reported pituitary obliteration

From the Department of Psychiatry (Dr. Brantley and Dr. Litovitz) and the Department of Medicine (Dr. Schachner), Fairfax Hospital and Georgetown University School of Medicine. Address correspondence to Dr. Brantley at Fairfax Hospital, 3300 Gallows Road, Falls Church VA 22046.



in a woman who died in a cachectic state. For many years thereafter, anorexic patients were frequently misdiagnosed as suffering from panhypopituitarism and treated ineffectively for that condition.

### Definition

The term "anorexia" is actually a misnomer, since patients often do not have a true loss of appetite. Rather, they fear weight gain or have a relentless pursuit of thinness. The term puberal-starvation-amenorrhea has been suggested as a more accurate name for the disorder.<sup>2</sup> The cardinal clinical features of anorexia nervosa are self-induced starvation, fear of losing control and becoming fat, and amenorrhea. In 1972, Feighner<sup>3</sup> developed diagnostic criteria for anorexia nervosa that avoided controversy over etiology or psychodynamic issues and deemphasized physical manifestations that could be secondary to starvation. Feighner set out the following criteria: 1) age of onset prior to 25; 2) weight loss of at least 25% of original body weight; 3) a distorted attitude towards eating or body weight, e.g., denial of illness with a failure to recognize nutritional needs, apparent enjoyment in losing weight, a desired body image of extreme thinness, and unusual hoarding or handling of food; 4) no known medical illness to account for the weight loss; 5) no other known psychiatric disorder; and, 6) the presence of at least two of the following problems: amenorrhea, lanugo, bradycardia, overactivity, bulimia, and vomiting (may be self-induced).

Some patients may ingest excessive amounts of food (binging) followed by vomiting, laxative abuse or diuretic abuse. Gorging with or without vomiting is called bulimia and characterizes a subgroup of anorexics. It is also found in normal weight and obese individuals. Bulimia may be a separate disorder or it may be on a spectrum with anorexia nervosa. Self-enforced hyperactivity and demanding exercise routines are commonly seen in both restricting and gorging groups of anorexics.

### Epidemiology

The onset of anorexia nervosa is generally between ages 10 and 25 with a peak onset in mid-adolescence, although occasional onset has been reported in children and postmenopausal women. Women are affected ten times more often than men and the majority of cases are among middle- or upper-class whites. The incidence of anorexia nervosa in the general population is approximately 1 per 100,000, but the prevalence among school-age girls may be as high as 1%-3%. A high incidence of

anorexia nervosa has also been reported in patients with Turner's syndrome and X chromosome monosomy. The incidence in post-pubertal girls has increased in industrialized countries in the last few decades. This increase is often attributed to changing societal attitudes and pressures towards thinness.

### Medical and Endocrine Features

Anorexia nervosa has many symptoms in common with involuntary malnutrition. However, the relative predominance of carbohydrate and calorie restriction over vitamin and protein restriction leads to some differences. Physical symptoms of anorexia nervosa include amenorrhea (nearly all cases), constipation or diarrhea (may be self-induced), abdominal discomfort, post-prandial nausea, cold intolerance, emesis, dry skin, brittle hair and nails, fatigue, insomnia and nocturia. On examination, patients appear cachectic and emaciated with striking loss of subcutaneous fat, although muscles are often well preserved. Other findings may include dry, scaly, and often carotenemic skin; lanugo hair; bradycardia; hypotension; acrocyanosis; hypothermia; decreased blood volume and heart volume; and peripheral edema. Patients often present to gynecologists because of amenorrhea, to gastroenterologists because of non-specific gastrointestinal symptoms, or to endocrinologists because of weight loss and fatigue. Abnormal laboratory findings include elevated BUN, low T<sub>3</sub>, decreased basal LH and FSH levels, elevated growth hormone levels, hypercarotenemia, and increased serum cholesterol levels.<sup>4</sup>

Hematologic changes in anorexia nervosa are usually mild and of minimal clinical significance. Bone marrow is markedly hypocellular and depleted of fat. Anemia (normochromic, normocytic) may be present in some but is mild, as is the leukopenia and thrombocytopenia. Erythrocyte acanthosis and poikilocytosis is common, probably related to reduced beta lipoprotein levels in these patients. Serum iron, iron binding, folic acid levels, and B<sub>12</sub> levels are almost always normal. Unlike other forms of malnutrition, immunologic function appears to be intact.

A number of cardiovascular effects have been noted in patients with anorexia nervosa. Heart rate and blood pressure are decreased. Blood volume and heart volume (by echocardiography) are decreased in proportion to the degree of weight loss as is seen in other forms of malnutrition. The EKG may show low voltage with flat or inverted T waves, prolonged QT interval, ST segment

changes, and ventricular ectopy. It has been suggested that the abnormalities in cardiovascular function correlate with the decreases in plasma norepinephrine concentration noted in these patients.<sup>5</sup>

Renal dysfunction may include fluid retention, reduced glomerular filtration or impaired concentrating ability.<sup>4</sup> Serum urea nitrogen is often increased with serum creatinine usually normal. Emesis, diarrhea or diuretic abuse may complicate the prerenal azotemia, making this a most serious medical complication. Other metabolic complications of anorexia nervosa include hypokalemia, hypochloremia, and metabolic alkalosis. Electrolyte abnormalities are related to the peculiar behaviors of vomiting and purgative, laxative, or diuretic abuse rather than to the malnutrition itself. Hypoalbuminemia is less common than in other forms of starvation. Indeed, serum albumin may be normal or increased in anorexics. Findings of raised serum cholesterol and carotene along with normal or increased albumin may help to distinguish anorexia nervosa from other cachectic states in which these levels would be normal or low.

Gastrointestinal function is relatively unimpaired in anorexia nervosa. Liver function tests, serum calcium, and fecal fat are usually normal although serum amylase may be elevated. Glucose tolerance tests range from flat to diabetic. Slow gastric emptying and esophageal dilatation have been noted in some. Patients frequently complain of constipation and dull to crampy abdominal pain, but upper and lower GI series are normal.

A great deal of research has focused on the endocrinologic aspects of anorexia nervosa with speculation as to whether hypothalamic dysfunction is primary or secondary to the starvation. However, it has been shown that starvation by itself can produce the same endocrine effects noted in anorexia. Nearly all women with anorexia nervosa have either primary or secondary amenorrhea. In men, libido and spermatogenesis are diminished. Amenorrhea begins prior to the onset of dieting or weight loss in over a third of cases and may persist despite return to normal weight. It has been suggested that amenorrhea may be related to psychogenic features in some patients and to weight loss in others.<sup>4</sup>

A selective deficiency in the secretion of pituitary hormones is found in anorexia nervosa.<sup>2</sup> Plasma levels of luteinizing hormone (LH), follicle stimulating hormone (FSH), and estradiol are decreased, and the circadian pattern of gonadotrophin secretion regresses to a prepubertal level. Nocturnal

FSH and LH surges characteristic of midpuberty and increased daytime surges of FSH and LH characteristic of later puberty are absent. Peak LH and FSH responses to gonadotrophin releasing hormone (GnRH) are also low, but repeated injections of GnRH correct this phenomenon, indicating a diminished pituitary responsiveness after long term absence of stimulation by GnRH. Thus, the gonadotrophin deficiency is hypothalamic rather than pituitary in origin. With restoration of weight, the gonadotrophin secretory pattern returns to an adult one in many patients.

The clinical picture of anorexia nervosa might suggest either hyperthyroidism (hyperactivity) or hypothyroidism (constipation, dry skin and cold intolerance). Thyroid studies in series of patients have shown normal T<sub>4</sub> and free T<sub>4</sub> levels with mild hypotriiodothyroninemia (low T<sub>3</sub> syndrome) in some.<sup>4</sup> Thyroid stimulating hormone is usually normal. It has been suggested that low T<sub>3</sub> is a common feature in many chronic illnesses, malnutrition, or fasting. The mechanism seems to be a decreased hepatic conversion of T<sub>4</sub> to T<sub>3</sub> or increased conversion of T<sub>4</sub> to the inactive isomer, reverse T<sub>3</sub>. Patients' failure to show normal thermoregulatory responses to heat or cold stress with a prominent absence of shivering reflects hypothalamic dysfunction. The greater the weight loss, the more deviant the response.<sup>2</sup>

### Psychological Features

Many anorexia nervosa patients fit a "typical" personality profile of obsessive-compulsive behavior with excessive concern for neatness, order, control and conformity. They are often described as having been extremely well-behaved, model children with superior school performance coupled with unrealistic fears of failure. Patients frequently come from upper middle-class, achievement-oriented families in which the parents are overly ambitious, perfectionistic and preoccupied with outer appearances, e.g., physical fitness, beauty and achievement. Frequently the mothers are over-directive and fail to acknowledge their children's individuality, thereby creating a climate where emotional growth is not promoted. Children reared in such an atmosphere often have fragile, low self-esteem that is overly dependent on approval from others.<sup>4</sup> A major psychological feature of anorexics is a "paralyzing sense of ineffectiveness".<sup>6</sup> Other features include depression-proneness, irritability, phobias, magical thinking, shyness, ritualistic behavior, sexual immaturity and naiveté.

Patients tend to overestimate their body widths to



a significantly higher degree than do non-anorexics. A postulated psychological mechanism for this body image distortion, which is found in virtually all anorexics, has been described by Bruch. She postulates that the body image distortion stems from inappropriate maternal responses to the infant's early communicative signals which leads to an impaired ability to accurately discriminate various bodily sensations, including hunger.<sup>6</sup> Psychological tests show a personality profile of high neuroticism, introversion, obsessiveness, as well as rigid conformity to the perceived desires of parents.

### **Etiology**

There is no consensus about the etiology of anorexia nervosa. Much research and attention has been directed at uncovering a physiological cause of the disorder. However, to date, little substantial scientific evidence has emerged to support the hypothesis of a primary physiologic disturbance. Rather, the physical changes accompanying the disorder are felt to be secondary to the observed malnutrition and are nearly always reversible when normal weight is attained.

Anorexia nervosa is most likely psychogenic in etiology and is often preceded by psychologically traumatic events, particularly injuries to a patient's self-esteem. The most common presentation involves a teen-age girl, perhaps slightly overweight, who is teased or criticized about her size and goes on a strict diet but is unable to stop dieting after having lost the initially desired amount of weight. She may insist that she looks and feels fat and will continue her diet on that basis.

After years of having felt generally ineffective, anorexics often experience a kind of exhilaration following a successful diet. They typically have had difficulty controlling their impulses and often express a fear that if they relax their vigilance even slightly, they will totally lose control and eat themselves into fatness. They tend to demonstrate this kind of all-or-none thinking in other areas as well. They may imagine that by achieving extreme thinness they will receive the approval and admiration which, because of a defective inner capacity to regulate and maintain self-esteem, they require for psychological equilibrium. As in most psychiatric disorders, many underlying unconscious factors converge to produce the manifest syndrome, self-starvation in this case. Anorexics often have an inadequate sense of identity and a sense of falseness of personality. They come to identify with their bodies and the discipline, structure and control that their bodies represent. This identification gives

them a feeling of being a "somebody." For the first time they may lose the sense of being a "nobody."<sup>6</sup> Other factors include anxieties related to impending parental separation, adult responsibilities, independence, and sexuality. The anorexic symptom may also represent an expression of anger toward parents, a concrete substitute for feeling in control of unacceptable sexual and aggressive drives, as well as a way of controlling parents, particularly when relations between parents are strained.

### **Treatment**

Hospitalization is indicated if weight is severely lowered or rapidly declining, if serious metabolic abnormalities are present, if the patient is suicidal, or if competent outpatient treatment has failed. If none of these conditions exists, outpatient treatment may be attempted. This can be accomplished by the primary care physician, who should see the patient frequently with a persistent focus on diet, weight and psychological concerns. However, successful outpatient treatment often requires fairly intensive psychotherapy, usually requiring referral, ideally to a psychiatrist with special interest and experience in eating disorders. If hospitalization is chosen, it is best provided on a psychiatric unit, although it can be accomplished on a general medical or pediatric ward. The generally accepted program involves a combination of behavioral techniques and psychotherapy. The former may not be necessary if the patient is cooperative and will gain weight according to schedule. This can be facilitated by the patient's being assigned a primary nurse or nurses who foster a trusting relationship with the patient and closely supervise her eating activities as well as reward her for weight gain with praise and encouragement.<sup>7</sup> However, the more typical patient denies her illness and requires behavioral contingencies for successful weight gain. A negative reinforcement plan (escape from an unpleasant situation being the stimulus for the desired behavior, i.e., eating and weight gain) calls for complete bedrest and lack of hospital privileges until weight gain is achieved.<sup>8</sup> Other behavioral regimens do not require bedrest but reward eating and weight gain with increased activities, television time, passes, etc. Additional measures, including drug treatment, e.g., neuroleptics, antidepressants, appetite stimulants, as well as electroconvulsive therapy, have been tried with mixed and inconsistent results. Now hyperalimentation is available for intractable patients and early treatment results appear very promising. Total parenteral nutrition, combined with psychotherapy, has been recommended for patients

who have failed to respond to other approaches, have life-threatening weight loss with marasmus and low potassium, or have increasing apathy, social isolation and depression. Contraindications for this regimen include a likelihood of manipulation of the IV and an inability to collaborate with the concomitant psychotherapy.<sup>9</sup> Of course, appropriate pharmacological treatment is indicated if there is a concurrent psychiatric disorder, e.g., major affective disorder, schizophrenia, and other psychoses. Hospital treatment should include frequent individual psychotherapy focusing on the patient's underlying feelings of inadequacy and ineffectiveness, fears of bodily changes, sexual and aggressive urges, conflicts within the family, and any other significant related psychological issues.<sup>10</sup>

Family therapy is often essential for effective treatment, especially when disturbed relationships exist within the family and are related to the patient's symptoms.

No special diet is required for refeeding. Because of the possibility of circulatory overload and stomach dilation, the patient should be started out with approximately 1,500-2,000 calories a day in six equal feedings.<sup>11</sup> After a week, as much as 3,000-5,000 calories/day can be consumed. Concentrated high calorie supplements may be used. Patients should be weighed daily after emptying their bladders.

In general, followup outpatient psychotherapy should be arranged, not only for the purpose of maintenance and prophylaxis, but for treating the invariably present underlying psychological problems that impair the patient.

### Prognosis

The course and prognosis of anorexia nervosa is highly variable, ranging from complete recovery with or without treatment to a progressive, downhill course with death resulting from starvation. Intermediate to these are fluctuating states of remissions and relapses, chronic but stable states of low body weight and anorexic behavior, and shifts to bulimia as a means of controlling weight.

The short-term response to hospital treatment is generally excellent, with improvements in nutrition, body weight and psychological functioning, especially that which is secondary to poor nutrition. However, long-term prognosis is less favorable. A recent review of 12 major outcome studies over the past 15 years showed that about half had shown complete recovery, 31% experienced some improvement in weight, and 18% did not change significantly. There was a combined mortality-mor-

bidity rate of 56% for anorexic problems alone, and a high percentage remained symptomatic in psychosocial areas. For example, of the nearly 500 patients for whom data was available, only 55% married or had dated significantly.<sup>12</sup>

Mortality from anorexia nervosa ranges in studies from 5%-21%. However, with more effective current treatment techniques, mortality from starvation can nearly always be averted. Studies of indicators of prognosis show early onset, good pre-morbid psychosocial adjustment, and less severe weight loss to be associated with good prognosis. Associated with poor prognosis are later age of onset, poor pre-morbid adjustment, more extreme weight loss, presence of family conflict, co-existence of bulimia and laxative abuse, and associated character psychopathology, e.g., significant hysterical, obsessive-compulsive and sociopathic features. In general, long-term prognosis, including psychological and social adjustment, varies according to the degree of underlying personality disorder.

In conclusion, it can be seen that anorexia nervosa is a complex syndrome which requires a combined medical-psychiatric approach for optimal treatment.

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# Bulimia

Kevin Kiernan, MD, Henry Dove, MD,  
and Thomas N. Wise, MD,  
*Falls Church, Virginia*

**B**ULIMIA, literally "ox-hunger", is an eating disorder characterized by episodic compulsive eating followed by self-induced vomiting.<sup>1</sup> This syndrome, frequently associated with other psychiatric disorders, such as anorexia nervosa, may be increasing in prevalence.

## Clinical Characteristics/Case Reports

The syndrome occurs most commonly in adolescents and young adults. A recent study noted that 19% of women and 5% of men in a college student population exhibited bulimic symptoms.<sup>2</sup> The clinical characteristics are ravenous consumption of food on an episodic basis, which is relieved by self-induced vomiting. The binge-purge behavior may be seen on a regular basis or only when the individual is under stress. The actual binge episode may allow consumption of up to 50,000 calories per day and may last for a few days.<sup>3</sup> Other individuals will binge rapidly over a period of several minutes. Binge episodes are often followed by feelings of guilt, depression, and purging. A persistent feeling of being overweight often promotes diuretic and laxative use in addition to self-induced vomiting. In contrast to individuals with anorexia nervosa, the bulimic individual often does not become emaciated and maintains a normal weight.

Bulimic individuals are often described as having outgoing personalities, but may admit to basic insecurity about their sense of attractiveness and sexuality.

*Case 1. A 17-year-old high school student of normal weight sought psychiatric help to stop her pattern of binge-eating, then vomiting.*

*She described herself as an anxious person who found that self-induced emesis following bingeing promoted a sense of relief. She admitted that maintaining her weight was also a concern that*

*made such purging acceptable. This behavior began in early adolescence, was greatly exacerbated when she began going steady with a boyfriend who made her feel socially restricted. In psychotherapy she improved when she was able to distance herself from her boyfriend and sought out other social activities with school-related clubs. She further improved when she was started on an antidepressant, imipramine.*

This case illustrates how the adolescent with vulnerable themes of sexuality and peer-related insecurities can present with symptoms of bingeing and purging. In this case, psychotherapy and antidepressant medication were helpful in modifying the difficulty. Emotional lability and impulsivity, manifest by suicide attempts and self-mutilation, are common. Kleptomania and substance abuse are often found in the history of individuals with bulimia.<sup>4</sup> Physical manifestations include parotid enlargement, rectal bleeding, alopecia, dental enamel loss, inflamed throat, and scars on the dorsum of the hands as well as esophageal tears.<sup>5</sup>

*Case 2. A 54-year-old woman was admitted for evaluation of gastrointestinal bleeding. On initial evaluation, the patient gave no history suggestive of a cause for her melena and underwent a rigorous investigation, including upper and lower gastrointestinal radiography and full laboratory evaluation. Endoscopy, however, revealed esophageal tears. Only then did the patient tearfully admit that she had experienced binge-eating episodes followed by self-induced vomiting since early adolescence. This behavior was never discovered. The patient had a lifelong history of fears of becoming overweight but had always maintained a normal weight. Diagnosed as bulimic, the patient entered psychotherapy and was able gradually to diminish this behavior.*

Laboratory examinations often reveal electrolyte disturbances. Hypokalemic alkalosis and EKG abnormalities often reflect such electrolyte abnormalities induced by vomiting, laxative and diuretic abuse.<sup>6</sup> Medical emergencies such as gastric rupture have been described, as has cardiac arrest from hypokalemia.<sup>6</sup>

## Differential Diagnosis

Some illnesses can mimic the physical manifestations of bulimia and need to be differentiated. In Kleine-Levin syndrome, which usually afflicts male adolescents, several times per year patients have episodes of alternating hyperphagia and hypersomnia.<sup>1</sup> In Kluver-Bucy syndrome, afflicting predominantly women, hyperphagia is also seen, usually in

connection with either hypo-or hyper-sexuality.<sup>1</sup> These patients have visual agnosia, reactivity to visual stimuli, and characteristic abnormal EEGs. Bartter's syndrome often presents in children with hypokalemic alkalosis.<sup>6</sup> This is secondary to juxtaglomerular cell hyperplasia with secondary hyperaldosteronism. Usually elevated renin or angiotensin levels are seen.

### Etiologic Factors

Sociocultural, psychological and physiologic etiologies have been invoked to explain bulimia. Society's prohibition against being fat has been noted to explain such behavior, whereas others feel that individuals with borderline personality disorders utilize the binge-emesis cycle as a means of coping with unexpressed hostility.<sup>7</sup>

Biologic explanations have pointed to the neurologic and endocrinologic systems. EEG abnormalities are often present in bulimics, but their exact meaning is unknown. Endocrinologic evaluations have demonstrated abnormal dexamethasone suppressant tests in a number of bulimic patients, which may relate to their relationship with major depressive illness as well as dysfunction in the hypothalamic-pituitary axis.<sup>8</sup> This data has suggested that bulimia may be related to affective disorders. Studies have shown that bulimia frequently occurs concurrently with an affective illness.<sup>6-9</sup> Family histories of affective disorders are also increased among bulimics when compared with psychiatric controls.<sup>9</sup>

### Treatment

The definitive treatment for bulimia remains unknown because of the obscure origins of the disorder itself. Nevertheless, a variety of psychiatric treatments, both psychodynamic, behavioral and psychopharmacologic, have been utilized. Both individual and group psychotherapy utilizing psychodynamic themes which allow the bulimic patient to understand various personality conflicts that lead to a binge-purge syndrome have been reported to be helpful.<sup>10</sup> Behavioral treatment utilizing positive reinforcement for eating without emesis, a cognitive approach, or response prevention have also been described with good results.<sup>11</sup> Finally, pharmacologic treatment for bulimia has focused on the use of anticonvulsive medication, as well as antidepressants. Phenytoin sodium (Dilantin®) may have some beneficial effects, especially in individuals with abnormal EEGs, but the studies are inconclusive.<sup>12</sup> Increasing information is appearing suggesting that antidepressants may have a major therapeutic role

in modifying the bulimic syndrome. Although only a few controlled studies have been carried out, imipramine has significantly reduced the binge-eating purge behaviors of bulimic subjects.<sup>13</sup> MAO inhibitors have also been suggested as effective agents.<sup>14</sup>

### Conclusion

Bulimia, which has been called the "secretive syndrome", is becoming increasingly recognized as a common disorder. Promising pharmacologic interventions may modify this serious, at times fatal, disorder. The first step in treating such a condition, however, is recognition. For this reason, the physician must keep a high index of suspicion in individuals who exhibit any characteristic physical symptoms.

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# Anorexia-Bulimia: Case Report

Barry R. Berkey, MD,  
*West Springfield, Virginia*

**T**HE CAUSE and cure of anorexia and bulimia, prototypes of the eating disorder syndromes, remain an enigma for both researchers and clinicians. The literature in recent years is more crowded than ever with studies, ranging from ingenious research approaches and recommendations flowing therefrom to various clinical treatment approaches and their outcomes. Yet there is no solid evidence of any therapeutic breakthroughs in the last 50 years.<sup>1</sup> The absence of any long-term, controlled, statistically valid studies pointing toward an effective treatment for either anorexia or bulimia continues to leave a void for those who face the distressing task of working with these afflicted individuals.

Bulimia often comes to the physician's attention as part of an anorexia-bulimia complex; as such, the focus of therapy is influenced by which symptoms, bulimia or anorexia, are more debilitating. In the case reported here, it is the latter. Significant weight loss is the principal problem that forces intervention; when such is the case, even prominent bulimia symptoms slip into the shadows.

The generally agreed upon treatment for anorexia—while important and often lifesaving—seems to be centered around hospital care that is primarily dietary and clearly more palliative than curative, since a high percentage of individuals continues to experience eating problems with concomitant psychological and social impairment.<sup>1-2</sup> Hospitalization is aimed principally at restoring weight and is implemented when the patient is sufficiently emaciated to require that the responsibility for eating be temporarily removed from the patient and placed with the attending multidisciplinary hospital team. Treatment<sup>1-7</sup> involves feeding the hospitalized patient necessary nutrients as well as structuring a milieu that may include social skills training, psy-

chotherapy, family and group therapy, behavioral techniques, and various kinds of drug therapy.

For bulimics, when hospitalization is required, cessation of bingeing and purging is the primary therapeutic focus.

The large number and types of pharmacologic agents used, for both disorders and for both in- and out-patients appears to indicate the physicians' search for "something that will work" and includes many different antidepressant medications (most commonly the tricyclics but also monoamine oxidase inhibitors), trazodone, tranlycypromine, phenelzine, anticonvulsants, lithium carbonate, L-dopa, clomipramine, chlorpromazine and insulin, and others.<sup>8-14</sup>

## Case Report

Mrs. A, a 40-year-old married woman with three children, presented with multisymptomatic expressions of depression and free-floating anxiety. Her thin appearance was initially overshadowed by her mild kyphosis, her rapid gait, scanning gaze and general restlessness. She spoke rapidly and wore a serious, intense facial expression. Attributing her distress to chronic marital conflicts, she revealed a long-standing history of deep-seated feelings of isolation, inadequacy, worthlessness, difficulty making independent decisions, and generally negative and pessimistic expressions about all aspects of her life—personal, familial, social and occupational. Virtually unable to relax, she felt compelled to keep busy with "something productive." She saw herself as having value only as a mother, devoting most of her energy to her children. She verbalized suicidal thoughts that once they no longer need her, there will be no reason for her to live.

Although never overweight, the patient indicated that she began to see herself as fat from about age 10 or 12, when her breast development began. Menses started at age 13 and she felt "extremely embarrassed" about this, relating that "my mother told me very little about it because she was embarrassed about it." Eating was reported as becoming a problem at this time, but only in the sense that she could not control what she ate because her mother controlled it. She described her mother as efficiently portioning out food for each family member, never asking anyone how much or if they wanted a particular food, and that "you ate what was on your plate." When in college, the patient likened eating in the cafeteria to eating at home. "It was an extension of the same way I'd eaten at home. I ate what I was given."

There were instances of binge-eating as a teen-

Address correspondence to Dr. Berkey at 8328-C Trafford Lane, West Springfield VA 22152.

Submitted 2-25-84.

ager and intimations of having induced vomiting and using laxatives, but she "felt horrified and embarrassed at having to admit such sick behavior as a youngster."

Reporting that at age 13, "I had a horrible scene with my mother—if I would get blood on me . . ." She believed her mother felt she was "messy, dirty, disorganized, very inefficient, sloppy . . ." and she described herself as a "slob, unattractive, dumpy and bulky and fat" from age 12 on. She said she wished she could be thinner, especially in her breasts and legs. She did not like "getting thicker through the hips like a woman" (as opposed to a girl).

The first occasion of abnormal eating occurred at age 19, over a summer break from college. Her parents, who, she says, "never got along," temporarily separated. "It was decided that I would go with my father" to a distant city, where the patient worked 12- to 14-hour shifts as a waitress in a restaurant. During this three-month period, the patient's father would take her out to eat once a week, and the patient said she ate her only meal at that time. Prompted by feeling "bulky and fat for as long as I can remember," she stated she ate "very little" between the weekly meals with her father. She did not recall how much weight she lost ("maybe 20 or 25 pounds") but did not binge, vomit or use laxatives. She established no friendships and described that summer as tense and terribly lonely. After returning to college, she resumed her cafeteria-like eating pattern.

Following the birth of her first child in 1969, she "dieted" for the second time. Stating that she became fat only with this child (one of four pregnancies), she lost 40 pounds in six weeks—(150 to 110 pounds). Revealing that she "stopped eating lunch and breakfast totally," she had only one meal a day. That meal consisted of a few pieces of vegetables.

Although she referred to dieting and using laxatives in 1971, the third distinct major dieting episode followed the stillbirth delivery by cesarian section of a deformed fetus in 1975; she had a tubal ligation at the time of the section. She said, "I was more upset about the tubal ligation than I was about the baby. I wanted to hurt myself so badly, I couldn't stand it." (Self-inflicted wounds are a part of this woman's behavior—cutting her arms with glass or a knife, hitting her legs with metal objects and producing ecchymoses. She also experiences erotic pleasure from masochistic dreams where she is tortured and demeaned by men.) Details of the amount of weight lost were non-specific. Stating that she "may have gone to 105 or so," she added,

"I think I can weigh 80 pounds and still feel I need to be thinner." Some vomiting and laxative use, along with daily weighing, occurred at this time.

Currently, this woman has been eating one meal a week for several months; a typical meal consists of some of the following: half cup broth, coffee with milk, 3-4 celery sticks, a few bell pepper strips, and a bite of toast. She eats and drinks some of the following most days: coffee, diet sodas, one or two carrot sticks, teaspoon cocoa powder, one cracker, teaspoon jelly, one-half of one orange section, one sip fruit juice, piece of raw vegetable such as cauliflower, pepper, carrot or celery, teaspoon honey or peanut butter. She will binge-eat three or four times a month, mostly with ice cream (a quart or more) or chocolate (up to 3/4 pound), and induce vomiting up to 20 times while bingeing. She frequently uses laxatives in doses several times stronger than recommended on the label. Her weight is about 95 pounds.

She views herself as disgustingly fat, and verbalizes a pervasive fear of eating. When she does eat, she experiences anger and guilt. It is the anger and guilt that results in her escape to the bathroom as soon after eating as possible to induce vomiting. This, plus the frequent use of laxatives, adds to the compulsive-like ritual in her feeding behavior.

## Discussion

Prior to a patient reaching the dangerous edge of personal disaster, where hospitalization is required, no cohesive treatment plan is agreed upon and recommended by eating disorder specialists. It is not surprising, therefore, that the causes as well as the treatment of this elusive and refractory illness remain speculative. Crisp<sup>15</sup> outlines a treatment course based on his theory of an underlying mechanism of anorexia nervosa being a weight phobia, which is related to an avoidance of maturing and having to deal with the stresses of adulthood. Fairburn and others believe that behavior therapy, sometimes with other treatment, such as group therapy, holds particular promise for patients with anorexia nervosa with or without bulimia.<sup>1,6,15-17</sup> Because bulimia is frequently associated with anorexia nervosa<sup>18</sup> and because this adds another dimension to the syndrome, the confusion factors—both as regards etiology and treatment—rise further. The presence of depressive symptoms suggesting an affective disorder in anorexia patients, especially those with bulimic symptoms, has led to the extensive use of antidepressant medication, especially tricyclics.<sup>13,14</sup> The benefits reported from the various trials of different medications are incon-



sistent, although most investigators believe that antidepressants do have a role in treatment, especially with bulimia.

In the case of Mrs. A, desipramine (to which thioridazine 10 mg tid was added after four weeks), amoxapine and trazodone were used separately at therapeutic doses for several months each. With trazodone her mood improved, and she reported having more energy and an improved outlook toward life in general, but no appreciable change in diet or bingeing was noted.

The patient was seen in individual psychotherapy biweekly and in group therapy weekly. She was referred to an internist to exclude contributing physical illnesses as well as for monitoring her nutritional status for possible hospital care. Cognitive behavioral therapy was a part of supportive and limited insight-oriented individual psychotherapy. The behavioral techniques provided information, eating guidelines, and reinforcement aimed at assisting the patient to gain the feeling that she herself could control her eating, vomiting and purging. Group therapy provided a matrix for support with a focus on reducing stress by resolving interpersonal conflicts related to major marital problems.

Strongly motivated by her own misery, the patient attended every scheduled session for over 12 months, but her fear of gaining weight remained morbidly obsessional as did her vomiting/purging. Her self-disgust when she ate did not abate, and she maintained a negative attitude and anxious preoccupation with feelings of responsibility for everything and everyone around her.

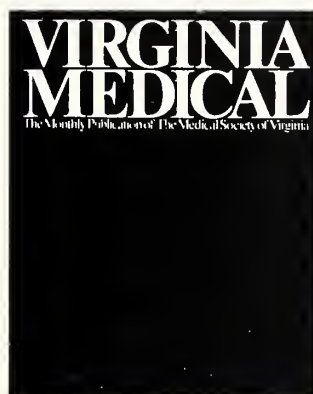
Treatment efforts in this case have included every reasonable modality based on sound theoretical implications, with the implementation of several techniques reportedly of benefit in others' cases.<sup>1,3-5,7,11,14-17</sup> Although some controlled studies with hopeful results are present, along with reports of improvement through the use of drugs, behavioral techniques and group therapy, fresh therapeutic directions are badly needed.

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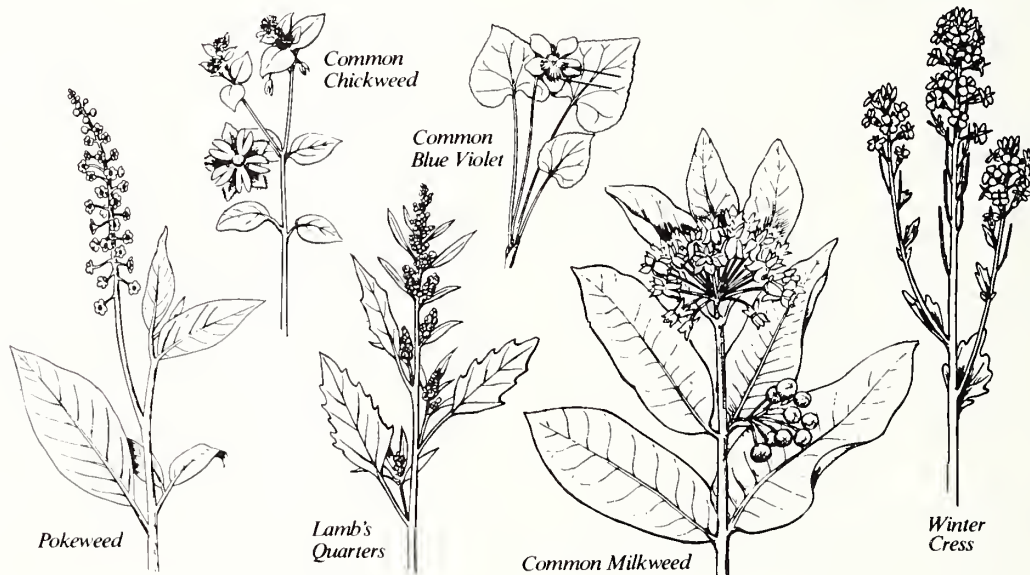
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# Foraging For Food

*I learned that a man may use as simple a diet as the animals and yet retain health and strength. I have made a satisfactory dinner off a dish of purslane which I gathered and boiled. . . . yet men have come to such a pass that they frequently starve, not for want of necessities, but for want of luxuries.*

—Henry David Thoreau



**F**EW OF US will ever be faced with the necessity of living off the land for an extended period of time, but in many of us the hunter-gatherer instincts still seek expression, and we enjoy foraging for food.

Virginia is a splendid place to pursue this satisfying pastime, for although modern-day life and its side effects have diminished the supply of wild food, there is still plenty of game for the hunters and plants for the gatherers.

Foraging for plants is my enthusiasm, and I have spent many invigorating hours in and around Leesburg, where I live, tracking down potherbs and greens for the family table. It is a hobby for which you need none of the expensive gear that characterizes the hunting and fishing crowd.

You do need an edible plant identification book, and a list of three good ones is appended. They will tell you what a plant looks like, where you may find

it, what part or parts of it are edible and which, conversely, are poisonous. They also provide information on the plant's season, when it is at its most palatable; many a forager has been turned off dandelions because they were picked too late and were bitter. A comprehensive book will also tell you how to prepare your forage for eating; by and large it takes more time and trouble than domesticated produce, but that's part of the experience, and you wind up with an uncommon dish.

Where to look, in Virginia, for edible plants? The likeliest places for foraging are abandoned farms, old fields, fence rows, beside country roads, around streams and ponds. Wilderness areas tend to be barren. Avoid busy highways, where plants may pick up toxic fumes exhausted by automobiles, and all areas sprayed with pesticides.

Foraging for edible plants is splendid for one's health, because of the walking and fresh air involved and because the plants are uniformly low in calories and high in vitamins.

Drawings from *A Field Guide to Edible Wild Plants* by Lee Allen Peterson. Copyright © 1977 Lee Allen Peterson. Reprinted permission of the publishers.



Here are six edible wild plants commonly available in Virginia.

### Winter Cress

The wild cress most commonly known to Americans is watercress (*Nasturtium officinale*), the succulent salad green offered in the summer by grocers. Quite a different plant is winter cress (*Barbarea vulgaris*), which is a member of the mustard family. As its name indicates, it may be gathered in late winter whenever the ground is free of snow—or even under the snow if you know where to look. Last March I cut over a bushel of winter cress from a field where soybeans had been harvested, in the settlement of Swampoodle near Leesburg. The young leaves, which form dense rosettes, are excellent either raw, as in a salad, or cooked, like other greens. Later on, when the leaves have become too bitter to use, tight clusters of flowerbuds appear; they can be cooked and served like broccoli.

### Chickweed

Chances are that all who garden in Virginia have pulled from flower bed or vegetable plot the common chickweed (*Sellaria media*). That the intruder was likely tossed onto mulch pile or rubbish heap is a pity, for the tender leaves and stems of the young chickweed add flavor and lots of vitamin C to salads. They may also be cooked, although for cooking one needs a larger quantity than is to be found in a carefully tended area and must go after foraging. My favorite source of chickweed is a vigorous stand of it along the walls of the church school building of St. Peter's Episcopal Church, where I am a parishioner.

### Poke

Poke, or pokeweed (*Phytolacca americana*), proliferates almost anywhere in Virginia—along roads, in cultivated fields and waste places. I can always find it in a grazing area at nearby Lovettsville called Polecat Plateau. A rank-growing plant reaching 6-8 feet tall, poke sends up from its perennial root each spring a crop of fat young shoots. Both leaves and stalks of these shoots are fine for the table, the leaves cooked like spinach, the shoots peeled and cooked like asparagus. But beware of poke's root, seeds, and mature stems and leaves; they are dangerously poisonous. When collecting the young shoots do not include part of the root. A plant variously called Indian poke or false hellebore (*Veratrum viride*) is notoriously toxic and should be avoided altogether.

### Lamb's-quarters

Both leaves and seeds of lamb's-quarters, or pigweed (*Chenopodium album*), have merit as edibles. The new leaves, cooked, are a favorite of my family. I pull the entire young plant, up to 4 inches high, then strip off the leaves and crown for boiling. Later on, the tender tips of the plant's many branches may be used in the same way. From the nutritious seeds one can make a hot breakfast cereal, my plant book tells me, although I have never tried it, nor have I ever ground the seeds into flour, as early Virginians did. Lamb's-quarters is very common in tilled areas, such as vegetable gardens; it comes along in late spring or early summer.

### Milkweed

As a general rule, poisonous plants are signalled by 1) milky sap or 2) red or white berries. An exception is the common milkweed (*Asclepias syriaca*); its milky sap, although mildly toxic, can be dispelled by boiling the young leaves, shoots and flowerbuds in several changes of boiling water. The plant's pods, when young and hard, can be prepared in the same way, and some fry the flowers in batter to make fritters. Milkweed grows in dry soil everywhere. I gather it easily in early summer in the fields and country roads near my home on Hogback Mountain, west of Leesburg.

### Violet

The beguiling violet is an edible plant. I gather the common blue violet (*Viola papilionacea*), picking the young leaves before the flowers appear. These green heart shapes can be cooked but are especially nice in a salad; either way they provide the eater with lots of vitamin A and C. Violet leaves can be mildly cathartic, however, so if you haven't eaten them before, try them first in small quantity. When the flowers appear, they, too, can be tossed into a salad.

—PRISCILLA L. FOOTE, MD  
with Ann Gray

Address correspondence to Dr. Foote at Route 2, Box 25, Leesburg, Virginia 22075

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### BRIEF SUMMARY

#### PROCARDIA® (nifedipine) CAPSULES

For Oral Use

**INDICATIONS AND USAGE: I. Vasospastic Angina:** PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation, 2) angina or coronary artery spasm provoked by ergonovine, or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

**II. Chronic Stable Angina (Classical Effort-Associated Angina):** PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

**CONTRAINDICATIONS:** Known hypersensitivity reaction to PROCARDIA.

**WARNINGS: Excessive Hypotension:** Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

**Increased Angina:** Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

**Beta Blocker Withdrawal:** Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

**Congestive Heart Failure:** Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

**PRECAUTIONS: General: Hypotension:** Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

**Peripheral edema:** Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

**Drug interactions:** Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates. PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis. Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility. When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy. Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

**ADVERSE REACTIONS:** The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

**Laboratory Tests:** Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

**HOW SUPPLIED:** Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77° F (15° to 25° C) in the manufacturer's original container.

More detailed professional information available on request

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**September 6-8**

**Doppler Echocardiography** (Wake Forest University/University of South Carolina), *Isle of Palms, South Carolina*. 24 Cat. I credits. Fee: Physicians, \$350; sonographers, \$250. Division of CME, 300 S. Hawthorne Rd., Winston-Salem NC 27103, (919) 748-4450.

**September 6-7**

**13th Annual Diagnostic Ultrasound in Obstetrics and Gynecology** (Johns Hopkins University), *Baltimore*. 16 Cat. I hrs. Fee: Physicians, \$240; technicians, \$165. Carlita Kearney, Office of CME, 720 Rutland Ave., Baltimore MD 21205, (301) 955-3168.

**September 8**

**Rheumatology** (Eastern Virginia Medical School), *Norfolk*. Office of CME, PO Box 1980, Norfolk VA 23501, (804) 446-5240.

**September 15-20**

**Annual Meeting, American Academy of Pediatrics**, *Chicago*. M. H. Jennison, MD, 1801 Hinman Ave., Evanston IL 60204, (312) 869-4255.

**September 15-20**

**Annual Meeting, American College of Radiology**, *Los Angeles*. R. W. Harris, Executive Director, 20 N. Wacker Dr., Chicago IL 60606, (312) 236-4963.

**September 20-23**

**Annual Meeting, American Society of Internal Medicine**, *San Antonio, Texas*. Barbara Lauter, 1101 Vermont Ave., NW, Washington DC 20005, (202) 289-1700.

**September 20**

**Use of Computers in the Medical Office** (East Tennessee State University), *Johnson City, Tennessee*. 6 Cat. I hrs. Fee: \$60. Sue Hutchinson, Office of CME, Quillen-Dishner College of Medicine, (615) 928-6426.

**September 21**

**7th Annual Perinatal Conference** (Medical College of Virginia), *Richmond*. 7 Cat. I hrs. Fee: \$90. Mrs. Beth Winn, Office of CME, Box 48, MCV Station, Richmond VA 23298, (804) 786-0494.

**September 22**

**First National Conference on the Triathlete** (Virginia Beach General Hospital), *Virginia Beach*. 8 Cat. I hrs. Fee: \$100. Clarke Russ, MD, 1016 First Colonial Rd., Virginia Beach VA 23454.

**September 24-28**

**In Vitro Fertilization** (Eastern Virginia Medical School), *Norfolk*. Office of CME, PO Box 1980, Norfolk VA 23501, (804) 446-5240.

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# VIRGINIA MEDICAL

## Weight Loss, Nutrition, and a Brochure

**W**EIGHT REDUCTION is a major personal concern for many Virginians. Whether that concern reflects health or cosmetic considerations, losing excess weight is an objective which can be very difficult to achieve. Diets for weight reduction have been proposed in books and popular magazines for years, a fact that indicates the failure of many "fad" diets that continue to appear at an increasing rate. In addition to the advice available through the popular press, an increasing number of weight reduction organizations have established programs in local communities.

The Nutrition Committee of the Virginia Council of Health and Medical Care decided to learn more about the weight control programs being conducted in Virginia. In the past year, several were invited to make presentations to the committee. As a result of this review, committee members established a subcommittee to find ways to provide information enabling the consumer to select a program geared to sound nutrition.

Health hazards the committee hoped to discourage included "crash" diets that provide extremely low calorie levels; diets that exclude entire food groups (often milk and grain products); and diets that emphasize a high protein intake with inadequate intake of other important nutrients. Problems with high protein diets not often recognized by the general public include the usually low fiber content and the negative calcium balance that may result from the associated increase in calciuria. Middle-

aged women vulnerable to osteoporosis who select a high protein diet may suffer double jeopardy: calcium loss via the urine compounded by reduction of dairy products in the diet.

The brochure offered in this issue, "Guidelines for Choosing a Weight Loss Program," is a product

### Edgar Fisher to Retire

About a year ago we published an editorial lauding the activities and accomplishments of the Virginia Council on Health and Medical Care headed so ably by Edgar J. Fisher, Jr. The Virginia Council is, of course, best known to the medical profession for its Physician Referral Service, a clearing house to bring together areas of the state needing physicians with physicians who are looking for a place to practice.

The Council has been extremely effective in other areas as well; for example, the weight-loss brochure in this issue was put together by its Nutrition Committee, as Dr. Patricia Hunt recounts in her editorial.

Now for the bad news. After 36 years as director, Edgar Fisher plans to retire in November 1984. The good work of the Council will continue, but Edgar Fisher and his leadership will be missed.

—E.L.K., JR.



of the Nutrition Committee's effort. Printed as a courtesy by Blue Cross and Blue Shield of Virginia, it is offered to physicians free of charge so that they may distribute it to patients for whom weight reduction is recommended. It is not intended for children or for pregnant or lactating women; their weight should be monitored only in a clinical setting.

Advice on nutrition is an integral part of health care. The Council's brochure was designed to help Virginia physicians as they provide that care.

PATRICIA A. HUNT, MD

Southside Virginia Training Center  
PO Box 4110, Petersburg VA 23803

## DRGs: Grim Realities, Many Questions

**I**F YOUR hospital's reporting period began on October 1, 1983, you have had contact with diagnosis-related groups (DRGs) under the new prospective payment system for Part A Medicare since day one. If not, you have just begun to feel the impact. The situation is still fluid, and any conclusions drawn now may well change. The final regulations were not published until January 1984, and those for the peer review organizations (PROs), the quality control arm of the system, were released more recently. Assessment of DRGs now defines a number of realities and raises perplexing questions.

The Health Care Finance Administration (HCFA) has stated repeatedly that physicians are responsible for the quality of medical care purchased in this prudent buyer approach, a role we readily accept. The warning comes from our colleagues organizing the Medical Society of Virginia Review Organization (MSVRO), who say that PRO will not have the equivalent capability or funding of the old Professional Standards Review Organizations. PRO scrutinizes only fringes and extremes; outliers (6%), short-interval readmissions, and a sample validation of the DRGs; big ticket items from a revenue-generating aspect, rather than broad quality assurance (94%). So we face quality assurance responsibility with limited means to accomplish the task, like building a house with a hacksaw and a ballpin hammer.

The most dramatic and attention-grabbing impact of this new reimbursement system has been the certification of the principal diagnosis. Physician reaction to this has been indignation at the implication of dishonesty, and the threat of criminal charge for suspected willful and deliberate falsification. The real cause for alarm may be the possible felony conviction for Medicare fraud, equivalent to billing Medicare for services not rendered. It is unclear whether or not the physician is certifying the coding, plus the principal diagnosis, and can the physician be responsible for the accuracy of the coding?

Accurate diagnosis is always good medical care, but the final diagnosis has never affected hospital income or threatened criminal action and thus carries a whole new meaning. The important thing is to recognize this new significance, and understand that the process begins at the moment admission is considered, since the principal diagnosis is based upon the reason for admission. Diagnostic workup planning and documentation of hospital course become more important than ever.

The fixed reimbursement for a given medical admission is the grim reality for hospitals. The DRG amount is *it*, and hospitals must make do or eat the overrun. We physicians must realize that hospitals are up against a financial wall.

So what we have is an adversarial situation between physicians and hospitals. Physicians face the long-term possibility of becoming the scapegoat of public reaction against any deterioration of quality of care and the short-term possibility of a felony charge if the principal diagnosis is challenged. The hospitals face a short-term threat of insolvency. Buried between these two powerful forces is the Medicare patient. Circling the perimeter of this struggle is the HCFA and the Federal Government. Sitting back watching it all are Congress and Industry. What a scene!

Cooperation between physicians and hospitals is mandatory now! No bullying, no threats and no arrogance. Administrators should resist the temptation to threaten loss of privileges if physicians do not cooperate in attesting to the "most profitable principal diagnosis possible." Physicians should not refuse to certify the front sheet on the grounds of self-incrimination. An atmosphere of mutual respect, patience and understanding of each other's responsibilities and problems must prevail. Balance and compromise must reign. Quality must take the edge over economy, but economy must be allowed wherever quality is not compromised.

The problem of quality assessment is one that no

one really wants to tackle. The complex and costly outcome audits under PSRO were probably the closest thing to real quality evaluation. Today, in the private sector, HMOs, PPOs and IPAs only state that quality assessment is being done, carefully avoiding specific details as to how.

What is the problem of giving the best care for the least money? Basically, it is knowing what happens when you do *not* do something. It is traditional medical research in the opposite direction. Medical research has always been the evaluation of an additional antibiotic, a new procedure, or a change in a therapeutic program aimed at improving outcome, reducing morbidity and mortality. No consideration at all was given to the increase in cost. Early post-MI or post-surgical ambulation was for a better result, and any economy that resulted was just fortunate spinoff. The DRG system is a program of retreat from this abundance. Now we must withhold or scale down care delivery in intensity and time without altering the effect on outcome. At this moment we are doing this purely intuitively. How do we measure effects in Medicare? By standard research techniques. We must design prospective studies, asking specific questions in controlled settings, looking for specific effects, just as we evaluate new medications or new surgical procedures. We should have been studying these effects before these economic restraints were enacted. What we are really doing is a massive empirical experiment blindly, and all we can do is wait and see what happens. This system is going to collect for us a massive volume of broad outcome data without any means to isolate these individual decisions, to determine under what circumstances they were made, or what options were available and the relationship between them. If the system breaks down (quality of care deteriorates according to this outcome data), we will not have any idea where the trouble is, or how to fix it.

Under the DRG system the hospital's Executive Committee of the Professional Staff (ECPS) now becomes the champion of the patient, of quality care, and of the individual rights of the staff physician. This point is emphasized when hospital administrators make it quite clear that the hospital legal staffs will not defend the physician against charges of falsifying the principal diagnosis. A vigorous and independent ECPS must protect the medical staff from undue pressures in the coding of the principal diagnosis, in the profiling of staff physicians, and in seeing that due process is carried out. One or two severely ill patients with prolonged hospital stays can markedly alter the average length

of stay profile of a physician, who otherwise may be doing well. In this example, should we be using average length of stay or median length of stay, or both, in evaluating physician performance? The ECPS should be responsible for understanding how physicians are being evaluated, monitoring physician's performance in the same way the hospital does and informing him of how he is doing before he is threatened with loss of staff privileges. Hospital administrators have always depended on the American Hospital Association and its state affiliates for help. The ECPS needs to follow this example, and look to the AMA's medical staff section for increased communication and support between organized medicine and hospital staffs. Medical staffs need to become separate, corporate bodies with their own administrative and legal staffs and independent finances.

As we venture further into DRGs, many problems may arise. The most currently evident one is getting from an accurate principal diagnosis to the ICD-9-CM coding to the appropriate DRG. This seems analogous to translating written word through three successive languages, English to Dutch to Greek. Is the medical records librarian responsible for this coding, which links the physician as the diagnosis originator with the appropriate DRG? Obviously, the hospital often stands to lose or gain hundreds to thousands of dollars in each event. The differences in reimbursement for the various types of pneumonia (\$2,940 to \$4,900) are examples of this. And guess who is under pressure in this situation? The problem of fitting our final diagnoses into the ICD code exists because medicine is an incredibly fluid science and the ICD is only a definition of terms at a given moment. Few of us have realized that records room librarians have been bending and squeezing our diagnoses to fit the ICD code for years. Now that this problem is center stage, physicians need to become more familiar with the codes so we can speak this language so critical to hospital reimbursement.

Somewhere buried in the center of all of this is the Medicare patient. What will the patient's reaction be to reduction in availability and quantity of medical care, in short, rationing? Time will tell us. If only the patient could be like the sun, in the center of our galaxy, getting all the attention the sun gets while exerting all the power the sun does. Who knows, the Medicare patient may do just this in the end. Maybe we had better stick with our patients.

H. C. ALEXANDER III, MD

2037 Crystal Spring Avenue, SW  
Roanoke VA 24014



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# VIRGINIA MEDICAL OBITUARY

## **Delos W. Boyer, MD**

Dr. Delos W. Boyer, Danville orthopedic surgeon, aged 63, died April 18 from injuries received in an automobile accident. Driving alone, he was en route to Nashville, Tennessee, when his car left the road and struck a concrete railroad trestle. He was on sabbatical from his orthopedics practice.

A native of Chicago, Dr. Boyer began his undergraduate education at Johns Hopkins University, Baltimore, and completed it at George Washington University, Washington, DC. He served from 1941 to 1944 in the US Air Force as a B-26 pilot, earning two Distinguished Flying Crosses. He was graduated from George Washington Medical School in 1950. He went to Duke University Hospital, Durham, for a residency in orthopedics, and soon after settled in Danville.

A member of the American Academy of Orthopaedic Surgeons and the American College of Surgeons, Dr. Boyer came to membership in The Medical Society of Virginia through the Danville-Pittsylvania Academy of Medicine.

## **Ben Steingold, MD**

Dr. Ben Steingold, for 45 years family practitioner in Norfolk, died April 4 in a Norfolk hospital. He was 76 years old.

A Norfolk native, Dr. Steingold was graduated from the University of Virginia and its medical school, Class of 1931. His internship and residency were served at Cumberland Hospital in Brooklyn and at Gouverneur Hospital in Manhattan, respectively. In 1934 he returned to Norfolk, never to leave again. Head of the Family Practice Department of Norfolk's Leigh Memorial Hospital for a time prior to that hospital's merge with Medical Center Hospitals, he was also a professor at Eastern Virginia Medical School.

Dr. Steingold's membership in The Medical Society of Virginia spanned 43 years, and he held membership also in the Norfolk Academy of Medicine and the American Academy of Family Physicians.

## **John S. Morris, Jr., MD**

Dr. John Sargent Morris, Jr., Lynchburg pediatrician for 34 years, died March 29 in Virginia Baptist Hospital, Lynchburg. He was 66 years old and had retired due to ill health.

Dr. Morris was a Lynchburg native and received his undergraduate education at Lynchburg College. He earned his medical degree in 1943 at the Medical College of Virginia and took his training in pediatrics at MCV and Johns Hopkins, Baltimore. He served for two years in the US Army Medical Corps before returning to his hometown in 1948 to establish a practice. Through his 23 years of service on the board of the March of Dimes, Dr. Morris was instrumental in establishing a genetics clinic in Lynchburg.

A longtime member of The Medical Society of Virginia, Dr. Morris was also a member of the Lynchburg Academy of Medicine, Virginia Chapter of the American Academy of Pediatrics and the Virginia Pediatric Society.

A son, Dr. Richard C. Morris, followed his father in the practice of medicine in Lynchburg.

## **Robley C. Allison, MD**

Dr. Robley Curtis Allison, Emporia family practitioner and for many years the area's medical examiner, died at home on May 4, at the age of 75.

Born and reared in Draper, Virginia, Dr. Allison was graduated from the College of William and Mary in 1932 and from the Medical College of Virginia in 1936. He trained at Central State Hospital, Petersburg, prior to World War II, when he served in the Army Medical Corps. Following his release from the service he returned to Central State and practiced there for two more years. In 1948 he established a practice in Emporia, where he was to remain for the rest of his life.

Dr. Allison came to membership in The Medical Society of Virginia over 45 years ago, through the Southside Virginia Medical Society. He was also a member of the American Academy of Family Physicians.

# Beyond the Thirtieth Day

You conclude that your patient should spend some time in a psychiatric hospital. More than a little time, given the nature of this particular problem.

The question is, where? Coming up with the best answer is seldom easy, for there are many factors to consider and many alternatives.

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Our approach is humanistic, so there is also a great deal of informal contact and ample opportunities for leisure activities, all carried out in a setting that provides warmth, comfort, privacy, beauty and as much freedom as possible. It is an environment conducive to healing.

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Contact Dr. David Waltos, Admissions Officer, Sheppard and Enoch Pratt Hospital, P.O. Box 6815, Baltimore, Maryland 21204. (301) 823-8200.



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# WHO'S WHO

## IN VIRGINIA MEDICINE

Coming up in Va Med's August issue, the classiest Who's Who of them all—a roster of members of The Medical Society of Virginia. It will be the second Membership Directory, successor to the first edition in 1982, and great care has been exerted to bring every member's name and address to an impeccable peak. (To the Editors' regret, the received requests for telephone numbers could not be honored for the simple reason that they are not programmed into the Society's computers.) In addition to the MSV membership, the issue will index for your reference the current officers of both component and specialty societies in Virginia.

These Medical Society of Virginia members have been elected to fellowship in the American College of Physicians: **Dr. Stephen L. Pohl**, Charlottesville; **Dr. Frederick C. N. Littleton, Jr.**, Kilmarnock; and **Dr. Stanley C. Tucker**, Richmond.

Beaver Creek Plantation, for two centuries home of Virginia's Hairston family, has been acquired by **Dr. J. Michael Bestler**, who has transformed it into a clinic for his cosmetic surgery practice. Now the City of Martinsville is close by, but in the beginning Beaver Creek was a King's grant of 30,000 acres of wilderness. It was given to Col. George Hairston, who built a brick plantation house on the site in 1776 and lived there until his death in 1827. When the original house

burned, it was replaced with a three-story wooden structure; revised and added to over the years, this house continued to domicile generations of Hairstons until title to the estate was transferred to Dr. Bestler.

The medical staff of Pulaski Community Hospital has elected **Dr. J. M. Hylton**, Pulaski family physician, as chief; at Loudoun Memorial Hospital in Leesburg, **Dr. Michael H. Willoughby**, Loudoun obstetrician-gynecologist, is the new chief of staff; and **Dr. Ralph M. Robinson**, surgeon, is the new chief of staff at the Fauquier Hospital in Warrenton.

At an alumni luncheon that was part of reunion activities at Davidson College, **Dr. W. T. Thompson, Jr.**, Richmond, received the Alumni Service Medal of the Davidson College Alumni Association. A member of Davidson's Class of 1934, Dr. Thompson has been a trustee of the college and president of its national alumni organization. He was for 15 years chairman of the Department of Medicine at the Medical College of Virginia and from 1976 to 1981 was Editor of VIRGINIA MEDICAL.

**Dr. George J. Carroll**, Suffolk pathologist and veteran secretary-treasurer of Virginia's State Board of Medicine, has been elected to the board of directors of the Federation of State Medical Boards of the United States.

In a trip sponsored by the United Jewish Federation of Norfolk, **Dr. Charles E. Horton** spent a week in Tel Aviv and Haifa, where he lectured and demonstrated techniques of plastic surgery at Sheba Medical Center and Tel Hashomer Hospital. Professor of plastic surgery at Eastern Virginia Medical School, Dr. Horton is also director of the

Eastern Virginia Graduate School of Medicine.

After 50 years as a family physician in Clifton Forge, **Dr. Walter E. Vermilya** retired early this year. He decided to call it quits, wrote a reporter for the *Daily Review*, because of a heart attack suffered in 1981 and a sore knee that was interfering with his ability to get around.

Dr. Vermilya's parents moved him to Clifton Forge from Greenville, New York, when he was one year old, and except for service with the Army Medical Corps in World War II, Virginia has been his setting ever since. He was graduated from Washington and Lee University, and went on to the Medical College of Virginia, where he was a freshman when an older brother, **Dr. G. Douglas Vermilya**, was in the senior class. Dr. Douglas Vermilya went on to practice surgery in Tazewell and is also now retired.

After internship in Raleigh, North Carolina, Dr. Walter Vermilya worked briefly for a coal company in Big Rock, Virginia, before setting up private practice in Clifton Forge.

**Dr. Walter Lawrence, Jr.**, director of the Medical College of Virginia's Massey Cancer Center and a member of VIRGINIA MEDICAL's Editorial Board, is one of the authors of the new *Manual of Soft Tissue Tumor Surgery* published by Springer-Verlag. The book deals with the treatment of soft tissue sarcomas, with emphasis on operative management. Dr. Lawrence's coauthors are Dr. James P. Neifeld, his associate in MCV's Division of Surgical Oncology, and Dr. Jose J. Terz, formerly of MCV and now at the City of Hope Medical Center, Duarte, California.

# VIRGINIA MEDICAL CLASSIFIED

*Virginia Medical classified ads accepted at the discretion of the Editor. Rates to Medical Society of Virginia members: \$15 per insertion up to 50 words, 25¢ each additional word. To non-members: \$30 per insertion up to 50 words, 25¢ each additional word. Deadline: 5th day of month prior to month of publication. Send to the Advertising Manager, 4205 Dover Road, Richmond VA 23221.*

**FACULTY PHYSICIAN**—Experienced family physician needed as full-time faculty director of Eastern Virginia Medical School Island Medical Center in Chincoteague, a fishing village and resort on scenic Eastern Shore. Practice staffed by third year FP resident. Medical students frequently on site. Coverage for vacation, CME. Other time off provided. Applicants must be board-certified FP's with successful practice experience, able to serve as clinical teacher and role model for residents and students. Administrative practice. Competitive salary and fringe benefits. Clinton H. Toewe II, MD, Chairman, Dept. Family Practice, EVMS, PO Box 1980, Norfolk VA 23501. Equal Opportunity Employer.

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**NEEDED**—Board certified or eligible FP physicians for recently constructed, newly equipped family health care center in Tidewater area. X-ray and lab included. Medical staff affiliation with medium-sized, well-equipped hospital. Beautiful, quaint community setting with population of 48,000 in three-mile radius. Average patient age 27. Guaranteed compensation with incentive plan. Coastal Health Services, 101 Buford Rd., #205, Richmond VA 23235, (804) 320-7549 or toll-free in Virginia, (800) 552-6638. Outside Virginia, (800) 551-1013.

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**PHYSICIAN** needed for full time position in new urgent care facility in Roanoke. Emergency medicine experience preferred. Salary \$60,000+. Malpractice and health insurance provided. Write: Emergency Medical Associates, Inc., PO Box 8622, Roanoke, VA 24014.

**NORTHERN VIRGINIA**—Fairfax. Part-time office space in beautiful new medical building minutes from Fairfax Hospital. Surrounded by 100 doctors. Ideal for internist, GP or any subspecialist. Call Dotty Smith at (703) 352-5861, immediate occupancy.

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## From the President: Who's Concerned?

**W**ho's concerned about what's happening to health care? I'll tell you who . . . the American doctor, you and me.

The cynic will say we're concerned because we might not make as much money as we used to. That, of course, could be partially true. But a greater truth is that many changes are upon us and no one knows where they will lead. Are we destined to have socialized medicine, which I don't think many American people really want, or something else? Is that something else going to be cheaper or more expensive? Is it going to mean better or worse care for our patients? Are we going to end up with outmoded hospitals and equipment, except for a few showplaces, as in Europe, or are we going to remain at the cutting edge in technology and hospital facilities?

These things are matters of great concern both for ourselves and our patients. After all, everyone is a potential patient, including us and our families. To what country will an American be able to go to get the latest in care, like the world now comes to the United States? Are we relegating our health care to a second class status because someone has decided that 10.5% of the gross national product is too much? Are these same "someones" some of the people who can afford to go any place any time because they have the wherewithall to do so?

Is our nation's health care destined to become a political issue to help a few politicians in the name of trying to help the poor and downtrodden? Are some of these individuals the same ones who know little about being poor or what the real problems of life are about? How many of these same people have

had a loved one critically ill whose life has been saved by new technology which is admittedly expensive?

There is no question that we are in the midst of a big change in medicine. Certainly, every effort should be made to make health care more cost effective, but cost cannot be the only factor. If it is, we may be in the position of throwing out the baby with the bath water. Remember how well government planners have helped our traffic problems and our educational system. Also think of the architects and engineers who have designed buildings, bridges and nuclear plants that have developed major problems. Also, let's not forget the economists and other financial wizards who always know just what to do to curtail inflation, interest rates and so forth, and are never heard from when their theories fail. These are the same types of people who are out to "help" and "improve" the health care of the people of this country and at a lesser cost. I am sure we have all heard that old saying, "We're from the Government and we're here to help you."

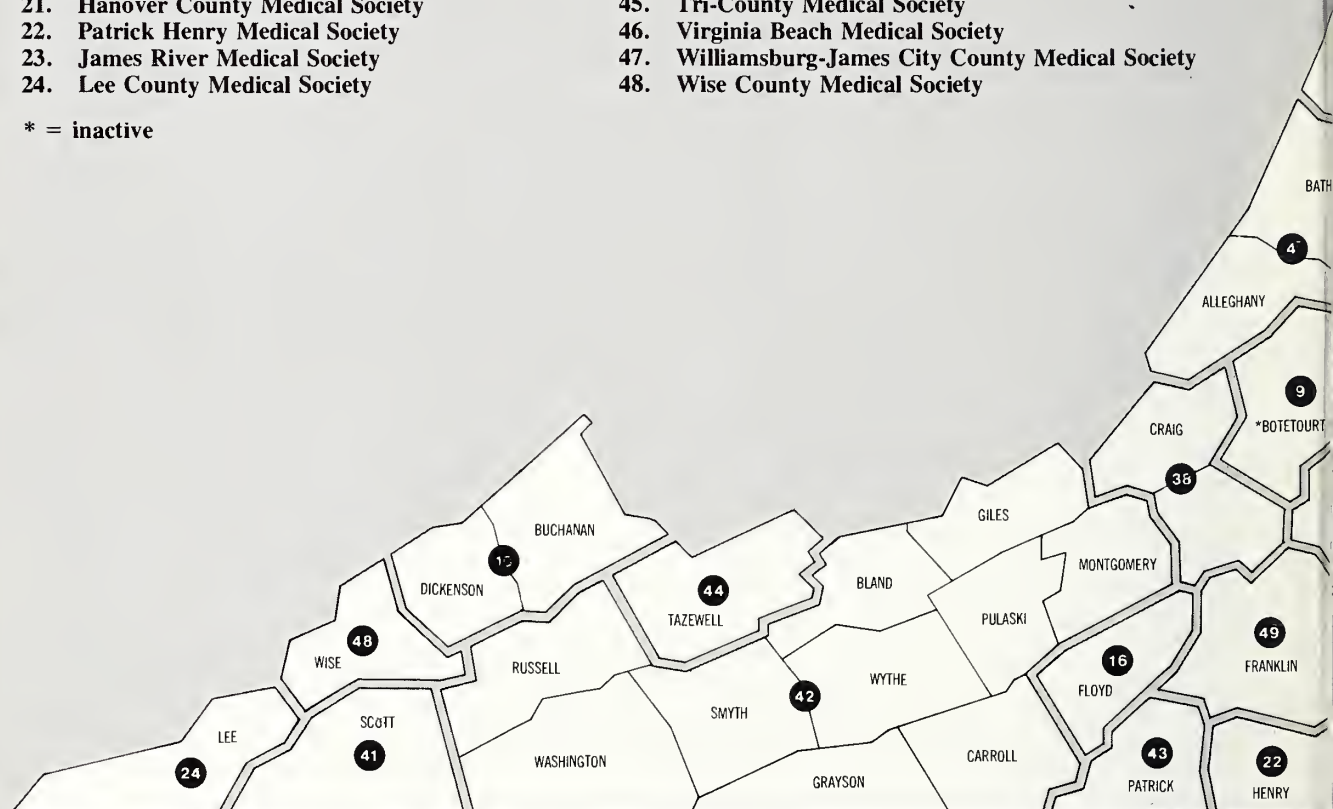
Indeed, it is clear that we physicians have good reason to be concerned. Someone must be concerned about quality. We are! This does not mean to say that the system is perfect and the costs should not be looked at. But in the process of improving the system, our nation must be careful not to injure it beyond recognition. The quality of our system of health care is a cause that American physicians must not only be concerned about but one that we must re-dedicate ourselves to leading.

**C. Barrie Cook, President,  
The Medical Society of Virginia**

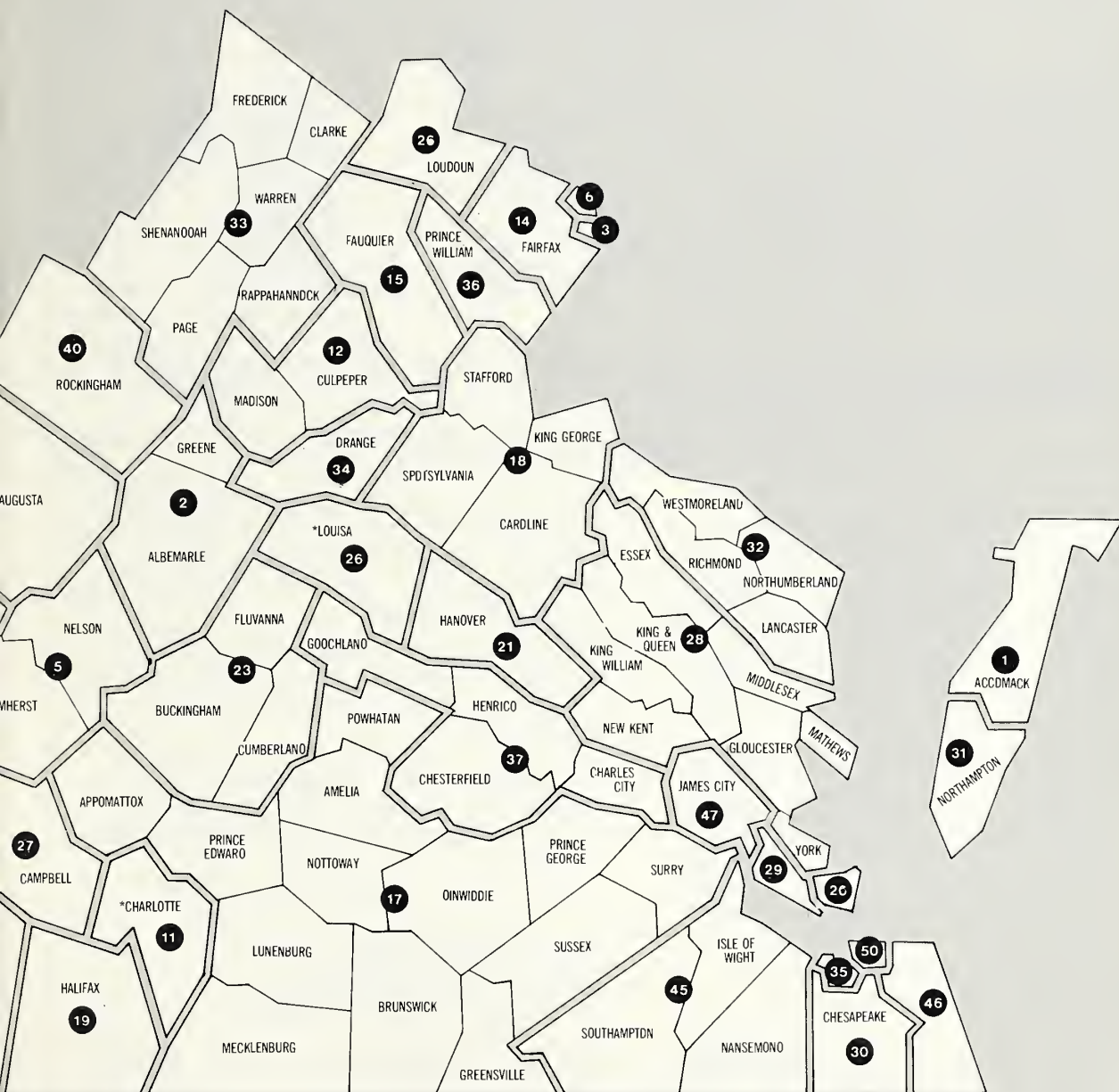
# County map showing geographical boundaries of Virginia's component medical societies

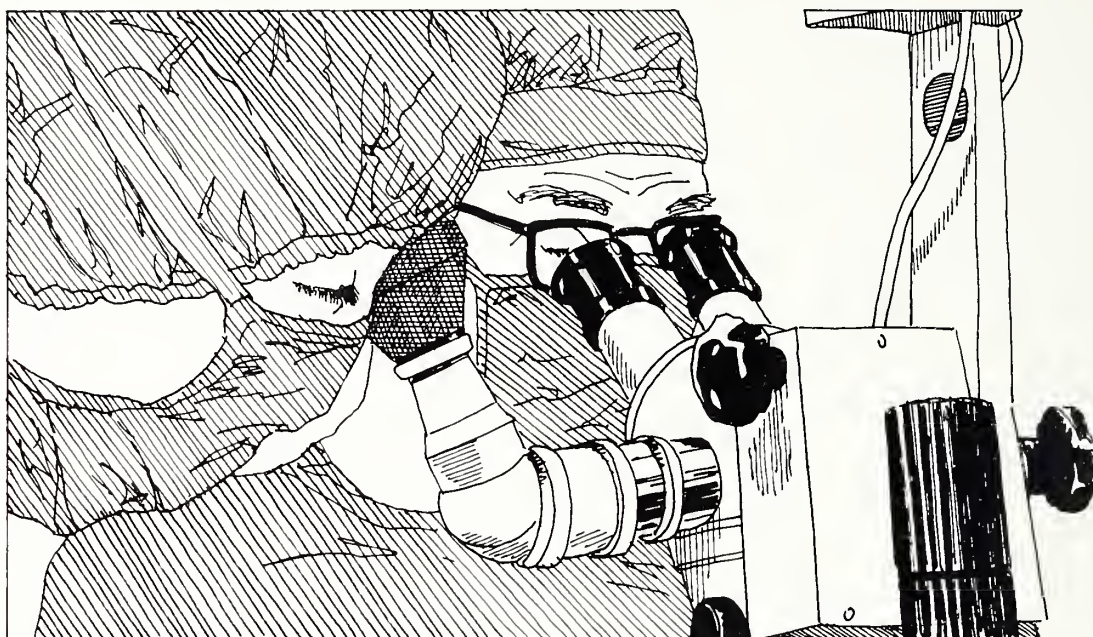
1. Accomack County Medical Society
2. Albemarle County Medical Society
3. Alexandria Medical Society
4. Alleghany-Bath Counties Medical Society
5. \*Amherst-Nelson Counties Medical Society
6. Arlington County Medical Society
7. Augusta County Medical Society
8. Bedford County Medical Society
9. \*Botetourt County Medical Society
10. Buchanan-Dickenson Counties Medical Society
11. \*Charlotte County Medical Society
30. Chesapeake Medical Society
12. Culpeper County Medical Society
13. Danville-Pittsylvania Academy of Medicine
14. Fairfax County Medical Society
15. Fauquier County Medical Society
16. Floyd County Medical Society
49. Franklin County Medical Society
18. Fredericksburg Medical Society
19. Halifax County Medical Society
20. Hampton Medical Society
21. Hanover County Medical Society
22. Patrick Henry Medical Society
23. James River Medical Society
24. Lee County Medical Society
25. Loudoun County Medical Society
26. \*Louisa County Medical Society
27. Lynchburg Academy of Medicine
28. Mid-Tidewater Medical Society
29. Newport News Medical Society
50. Norfolk Academy of Medicine
31. Northampton County Medical Society
32. Northern Neck Medical Society
33. Northern Virginia Medical Society
34. Orange County Medical Society
35. Portsmouth Academy of Medicine
36. Prince William County Medical Society
37. Richmond Academy of Medicine
38. Roanoke Academy of Medicine
39. Rockbridge County Medical Society
40. Rockingham County Medical Society
41. Scott County Medical Society
17. Southside Virginia Medical Society
42. Southwestern Virginia Medical Society
43. Stuart Medical Society
44. Tazewell County Medical Society
45. Tri-County Medical Society
46. Virginia Beach Medical Society
47. Williamsburg-James City County Medical Society
48. Wise County Medical Society

\* = inactive









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**VICE PRESIDENT**—E. W. Bosworth, MD, Onancock VA 23417  
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**EXECUTIVE SECRETARY**—Mrs. Betty H. Finn, Box 157, University of Virginia Medical Center, Charlottesville VA 22908

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**VICE PRESIDENT**—John W. Klousia, MD, 3323 Duke Street, #2-B, Alexandria VA 22314  
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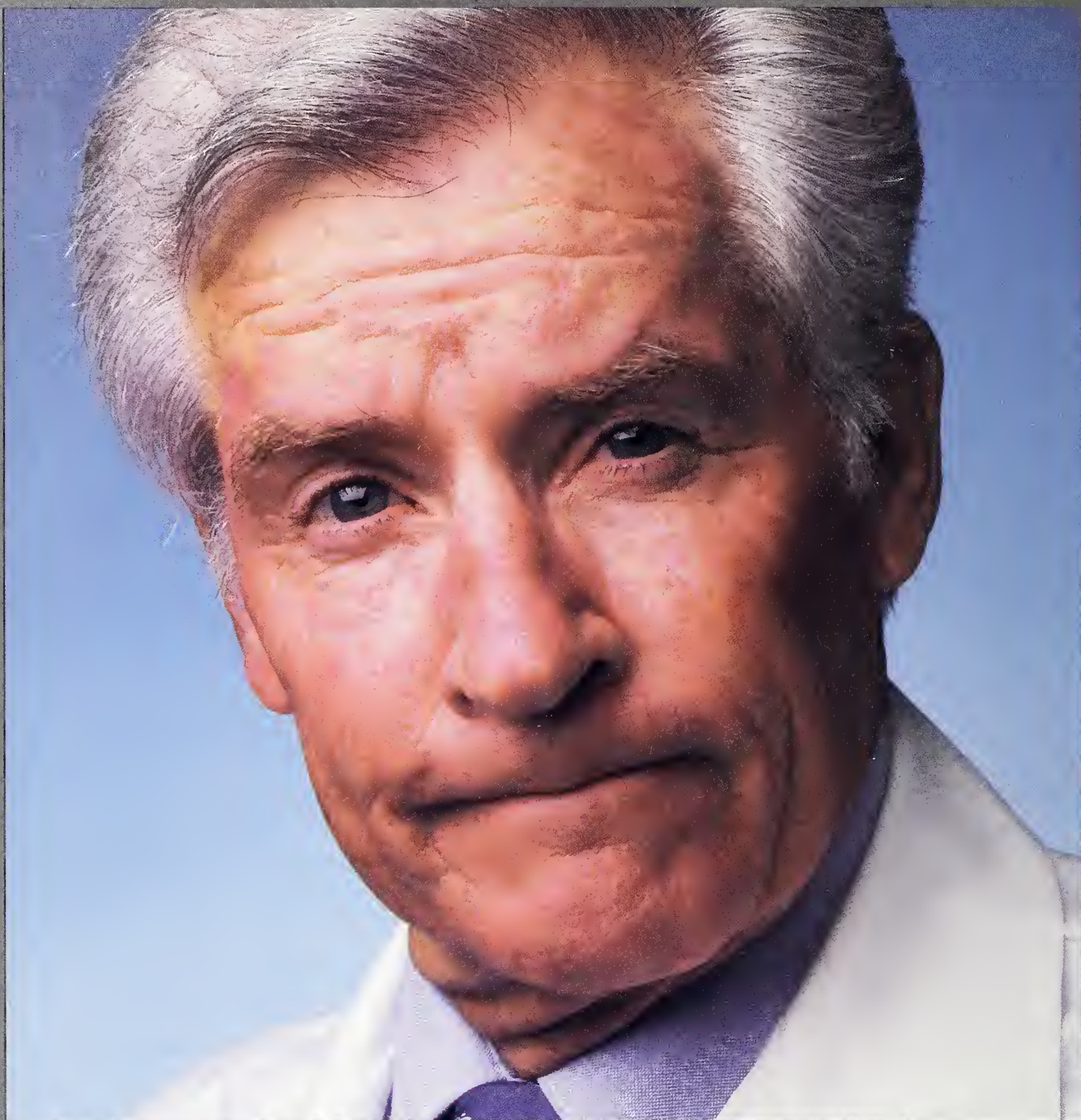
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**October 11-13, 1984**

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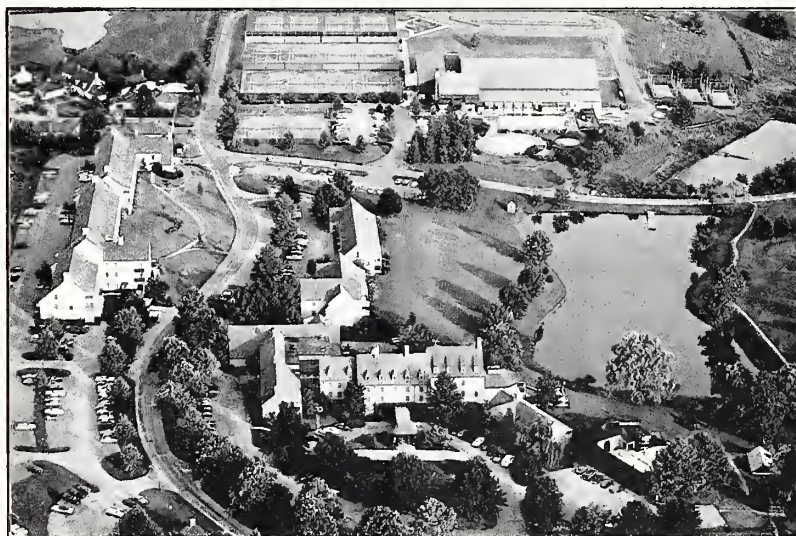
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<b>A</b> Allergy .....	36	<b>LM</b> Legal Medicine .....	2
<b>ABS</b> Abdominal Surgery .....	7	<b>ND</b> Neoplastic Diseases .....	10
<b>AM</b> Aerospace Medicine .....	5	<b>NEP</b> Nephrology .....	32
<b>AN</b> Anesthesiology .....	263	<b>N</b> Neurology .....	89
<b>CD</b> Cardiovascular Diseases .....	135	<b>NM</b> Nuclear Medicine .....	15
<b>CDS</b> Cardiovascular Surgery .....	23	<b>NS</b> Neurological Surgery .....	76
<b>CHN</b> Child Neurology .....	3	<b>NTR</b> Nutrition .....	1
<b>CHP</b> Child Psychiatry .....	68	<b>OBS</b> Obstetrics .....	1
<b>CLP</b> Clinical Pathology .....	16	<b>OBG</b> Obstetrics and Gynecology .....	450
<b>PA</b> Clinical Pharmacology .....	2	<b>OM</b> Occupational Medicine .....	28
<b>CRS</b> Colon and Rectal Surgery .....	11	<b>ON</b> Oncology .....	20
<b>D</b> Dermatology .....	109	<b>OPH</b> Ophthalmology .....	248
<b>DIA</b> Diabetes .....	3	<b>ORS</b> Orthopedic Surgery .....	293
<b>DR</b> Diagnostic Radiology .....	125	<b>OST</b> Osteopathic Medicine .....	14
<b>EM</b> Emergency Medicine .....	114	<b>OT</b> Otolaryngology .....	2
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<b>GE</b> Gastroenterology .....	65	<b>PDA</b> Pediatric Cardiology .....	9
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<b>GP</b> General Practice .....	492	<b>PD</b> Pediatrics .....	442
<b>GPM</b> General Preventive Medicine .....	8	<b>PM</b> Physical Medicine and Rehabilitation .....	30
<b>GS</b> General Surgery .....	450	<b>PS</b> Plastic Surgery .....	57
<b>GYN</b> Gynecology .....	44	<b>PYA</b> Psychoanalysis .....	9
<b>HS</b> Hand Surgery .....	6	<b>PYM</b> Psychosomatic Medicine .....	1
<b>HNS</b> Head and Neck Surgery .....	1	<b>P</b> Psychiatry .....	371
<b>HEM</b> Hematology .....	26	<b>PH</b> Public Health .....	44
<b>HYP</b> Hypnosis .....	1	<b>PUD</b> Pulmonary Diseases .....	55
<b>ID</b> Infectious Diseases .....	11	<b>R</b> Radiology .....	196
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<b>LAR</b> Laryngology .....	1	<b>TR</b> Therapeutic Radiology .....	26
		<b>TS</b> Thoracic Surgery .....	33
		<b>U</b> Urological Surgery .....	162
		<b>OS</b> Other .....	42
		<b>US</b> Unspecified .....	124
			6441
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			ANDREWS, MD, K Patricia M	AN		DAVIS IV., MD, John Staige	RHU		GUERRANT, MD, John L	IM	*
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			Charlottesville VA 22901			Charlottesville VA 22908			Charlottesville VA 22908		
			ARCHER, MD, Harry Lee	GS		DAVIS JR., MD, Ernest D	GP	*	KEATS, MD, Theodore Eliot	DR	*
			400 Locust Ave			Crozet VA 22932			Box 170, Uni Va Med Ctr		
			Charlottesville VA 22901			DAVIS, MD, Chas Monroe	P	*	Charlottesville VA 22908		
			ARMSTRONG JR., MD, Peter	US	*	Ri 1, Box 174-A			KEELING, MD, Richard Powell	IM	
			Box 170, Uni Va Med Ctr			Keswick VA 22947			Route 2, Box 111		
			Charlottesville VA 22908			DEE, MD, Paul Michael	R	*	Charlottesville, VA 22901		
			ARNOLD III, MD, William P	AN		Box 170, Uni Va Med Ctr			KELLY, MD, Thaddeus Elliott	OS	
			Box 238, Uni Va Med Ctr			Charlottesville VA 22908			108 Whatstone Place		
			Charlottesville VA 22908			DELAWARE, MD, Hilbert H	P		Charlottesville VA 22901		
			ASHLEY, MD, John T	PM	*	100 Court Square Annex, #1			KESLER, MD, Richard Wm	PD	
			Box 148, Uni Va Med Ctr			Charlottesville VA 22901			Box 386, Uni Va Med Ctr		
			Charlottesville, VA 22908			DERDEYN, MD, Andre Philip	CHP		Charlottesville VA 22908		
			ASKEW JR., MD, Fletcher C.	D		Box 202, Uni Va Med Ctr			KIM, MD, Jung-Ah Christina	TR	*
			1000 East High Street			Charlottesville VA 22908			Box 383, Uni Va Med Ctr		
			Charlottesville, VA 22901			DI FAZIO, MD, Cosmo Americo	AN		Charlottesville VA 22908		
			ATKIN, MD, Joan F.	EN		Box 238, Uni Va Med Ctr			KIRKLAND JR., MD, Nathaniel C	FP	
			Box 386, Uni Va Med Ctr			Charlottesville VA 22908			Doctors Office Building		
			Charlottesville, VA 22908			CARTER, MD, Bruce Thomas	OPH		Woodstock VA 22664		
			ATUK, MD, Nuzhet Osman	NEP		1101 E Jefferson St, #3			KISTNER, MD, James Robert	AN	
			7 Deeparth Road			Charlottesville, VA 22901			Box 238, Uni Va Med Ctr		
			Charlottesville, VA 22901			CATES, MD, Michael A.	OBG		Charlottesville VA 22908		
			AUSTIN, MD, Harry Paul	PD		1101 E Jefferson St, #4			KITCHIN III, MD, James David	OBG	*
			305 Barracks Hill			Charlottesville, VA 22901			U Va Hospital		
			Charlottesville VA 22901			CAUGHON, MD, Samuel Dan	EM		Charlottesville VA 22908		
			BAIN, MD, Thos Herbert	IM		211 East High Street			KNIGHT, MD, James Gregory	IM	
			Box 459			Charlottesville, VA 22901			400 Locust Avenue		
			Crozet VA 22932			CRAWLEY, MD, Edward Philip	D	*	Charlottesville VA 22901		
			BARNETT JR., MD, B Lewis	FP	*	Rugby Circle			KNORR, MD, Norman J	OS	*
			Box 414, Uni Va Med Ctr			Charlottesville VA 22903			Box 395, Uni Va Med Ctr		
			Charlottesville VA 22908			CHANDLER, MD, James Gilbert	GS	*	Charlottesville VA 22908		
			BARTON JR., MD, Chas Robt	GP		Box 181, Uni Va Med Ctr			KOVAC JR., MD, Michael John	ORS	*
			Route #3, Box 381			Charlottesville VA 22908			1101 E High St		
			Palmyra, VA 22963			CHESSLER, MD, David L.	IM		Charlottesville VA 22901		
			BEDFORD, MD, Robert Forrest	AN		2621 Commonwealth Dr			KRON, MD, Irving Louis	TS	*
			Box 238, Uni Va Med Ctr			Charlottesville VA 22901			Box 181 Uni Va Med Ctr		
			Charlottesville VA 22908			CHEVALIER, MD, Robert L	NEP		Charlottesville, VA 22908		
			BELLER, MD, George Allan	IM		Box 386, Uni Va Med Ctr			KULUND, MD, Daniel Nicholas	ORS	
			Box 158, Uni Va Med Ctr			Charlottesville VA 22908			503 Faulconer Drive, #3		
			Charlottesville, VA 22908			CLARK, MD, Mary Williams	ORS	*	Charlottesville VA 22901		
			BENDER, MD, Arthur Stillman	IM		2270 Ivy Road			LAKE, MD, Carol Lea	AN	*
			1011 East Jefferson St			Charlottesville, VA 22901			Box 238, Uni Va Med Ctr		
			Charlottesville VA 22901			CLARKE, MD, William Linus	PD		Charlottesville VA 22908		
			BENJAMIN, MD, John Tabb	IM		Box 386, Uni Va Med Ctr			LAMBERT III, MD, Matthew J	GS	
			24 West Leigh Drive			Charlottesville VA 22908			1100 E High Street		
			Charlottesville VA 22901			COATES, MD, Michael Lee	FP		Charlottesville VA 22901		
			BERK, MD, Barbara Zeviner	IM		Box 414, Uni Va Med Ctr					
			438 Wellington Drive			Charlottesville, VA 22908					
			Charlottesville VA 22901								

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LAMBERT, MD, Paul R. Box 430 Uni Va Med Ctr Charlottesville, VA 22908	OTO *	MOON JR., MD, Cary Nelson 1000 E High St Charlottesville VA 22901	OTO *	ROCHESTER, MD, Dudley F Box 225, Uni Va Med Ctr Charlottesville VA 22908	PUD *	TAYLOR, MD, Frank E 400 Locust Ave Charlottesville VA 22901	IM *	WYKER JR., MD, Arthur W Box 422, Uni Va Med Ctr Charlottesville VA 22908	U *
LAWSON, MD, Edgar Clifford 1011 E Jefferson St Charlottesville VA 22901	OPH *	MOONEY, MD, Joseph 1224 W Main St, #501 Charlottesville, VA 22903	P	RODGERS, MD, Bradley M. Box 181, Uni Va Med Ctr Charlottesville VA 22908	PDS	TEATES, MD, Chas David Box 486, Uni Va Med Ctr Charlottesville VA 22908	NM *	YQUEL JR., MD, John Kenneth 1011 E Jefferson St Charlottesville VA 22901	GS
LEAVELL JR., MD, Byrd S. 916 East High Street Charlottesville, VA 22901	GE	MORRIS III, MD, John R. 400 Locust Ave Charlottesville VA 22901	GS	ROGOL, MD, Alan David Box 386, Uni Va Med Ctr Charlottesville VA 22908	END	TEGMEYER, MD, Chas John Box 170, Uni Va Med Ctr Charlottesville VA 22908	DR *	ALEXANDRIA	
LEE, MD, Soo Ik Box 394, Uni Va Med Ctr Charlottesville VA 22908	N *	MORRIS JR., MD, John Richard 400 Locust Ave Charlottesville VA 22901	IM	ROSS JR., MD, Wm Tyler Box 238, Uni Va Med Ctr Charlottesville VA 22908	AN	THAGARAJAH, MD, Siva Box 387, Uni Va Med Ctr Charlottesville VA 22908	OBG	ABRAMSON, MD, Edward Gerald 1707 Osage St Alexandria VA 22302	U *
LEVIEU, MD, Michael G Box 386, Uni Va Med Ctr Charlottesville VA 22908	PD	MORRIS, MD, David L 400 Locust Ave Charlottesville VA 22901	IM	ROWLINGSON, MD, John Clyde Box 238, Uni Va Med Ctr Charlottesville VA 22908	US	THIELE, MD, Arthur L Martha Jefferson Hosp Charlottesville VA 22901	R	ACEVEDO, MD, Miguel A 2813 Duke Street Alexandria VA 22314	OTO
LINDSAY, MD, Richard Walter Box 157, Uni Va Med Ctr Charlottesville VA 22908	FP *	MORRIS, MD, J Leon Box 170, Uni Va Med Ctr Charlottesville VA 22908	DR *	RUDOLF, MD, Leslie Eugene Box 181, Uni Va Med Ctr Charlottesville VA 22908	GS *	THOMSON JR., MD, James A 1224 W Main St, #722 Charlottesville VA 22903	P	ADESON, MD, Robt Lawrence 5001 Seminary Rd Alexandria VA 22311	GS *
LLEDO, MD, Alfonso M. Box 170, Uni Va Med Ctr Charlottesville VA 22908	DR *	MORTON II, MD, C Bruce 8600 Midnight Pass, #28 Sarasota, FL 33581	GYN	RUSHA, MD, Edwin L 932 E Jefferson Charlottesville VA 22901	AN	THORNTON JR., MD, Wm N 134 Indian Spring Road Charlottesville VA 22901	OBG *	AHDOOT, MD, Habibollah 4701 Kenmore Ave, #119 Alexandria, VA 22304	OBG *
LOHR, MD, Jacob Andrew Box 386, Uni Va Med Ctr Charlottesville VA 22908	PD	MOSCA, MD, Alton Bernard 322 Westminster Rd Charlottesville VA 22901	IM	RUSHA, MD, Mary Anna 932 East Jefferson St Charlottesville, VA 22901	P	THORUP JR., MD, Oscar A Box 368, Uni Va Med Ctr Charlottesville VA 22908	IM *	ALEXANDER JR., MD, Edward G 5208 Dawes Ave Alexandria VA 22311	ORS
LONGNECKER, MD, David Eugene Box 238, Uni Va Med Ctr Charlottesville VA 22908	AN	MULLER JR., MD, Wm H Box 474, Uni Va Med Ctr Charlottesville VA 22908	TS *	SABIO JR., MD, Hernan Box 386, Uni Va Med Ctr Charlottesville VA 22908	HEM	TOMPKINS, MD, Dorothy E. Box 386, Uni Va Med Ctr Charlottesville VA 22908	PD	ALMASSIAN, MD, Hoosang 5258 Dawes Ave Alexandria VA 22204	OTO
LOONEY, MD, Wm Boyd Box 392, Uni Va Med Ctr Charlottesville VA 22908	OS	MURRAY, MD, Gary C. 1240 Nova Drive Waynesboro, VA 22980	CD *	SANDUSKY, MD, Wm Roberts 5 Canterbury Rd Charlottesville VA 22902	GS *	TOMPKINS, MD, William F. 1100 East Jefferson Charlottesville VA 22901	IM	ALPERSTEIN, MD, Joel Barry 1021 Seminary Rd, #230 Alexandria VA 22311	U
MAC ILWAINE, MD, Wm Andrew 820 East High Street Charlottesville, VA 22901	OPH	NELSON, MD, Douglas N. Box 134, Uni Va Med Ctr Charlottesville VA 22908	D	SANSONE, MD, Philip Andrew 2101 Arlington Blvd Charlottesville VA 22901	P *	TURNER III, MD, U.G. Box 378, Uni Va Med Ctr Charlottesville VA 22908	OBG	AMOLE, MD, Chas Varner Woodmere Drive, Apt 18E Petersburg VA 23805	GS *
MACFARLAN, MD, D. Andrew P O Box 197 Earlysville, VA 22936	FP *	NEWMAN, MD, Steven Alan 102 Oak Forest Cir Dr Charlottesville VA 22901	OPH *	SAULSBURY, MD, Frank T. 2585 Kimbrough Circle Charlottesville, VA 22901	PD	TWYMAN, MD, James Baker #3 Ednam Village Charlottesville VA 22901	IM *	ANDERSON, MD, Robt Harper 312 S Washington St, #2-C Alexandria VA 22314	PD *
MACKEL, MD, Susan E. 204 Pineridge Lane Charlottesville, VA 22901	D	NOLAN, MD, Stanton Paele Box 181, Uni Va Med Ctr Charlottesville VA 22908	CD *	SCHENK III, MD, Worthington G. Box 181, Uni Va Med Ctr Charlottesville VA 22908	GS	UNDERWOOD JR., MD, Paul B. Box 387, Uni Va Med Ctr Charlottesville VA 22908	GYN	ANDERSON, MD, Walter E 1904 Windmill Lane Alexandria VA 22307	CHP
MAGRUDER, MD, R. Gregory 3 Sunset Circle Charlottesville VA 22901	IM *	NUNLEY JR., MD, Wallace C. Box 387, Uni Va Med Ctr Charlottesville VA 22908	OBG	SCHILDWACHTER, MD, Thomas L 912 East High Street Charlottesville VA 22901	ORS	VAN DER SOMMEN, MD, Lynda D. P. O. Box 947 Louisia, VA 23093	GP	ANIXTER, MD, William L 1444 Duke Street Alexandria, VA 22314	P *
MARSHALL JR., MD, Hubert A 408 E Market St, #104 Charlottesville VA 22901	OPH *	OBLINGER, MD, Michael J. 916 East High Street Charlottesville VA 22901	GE	SEALE, MD, Danl Logan Rt 7 Box 169 Charlottesville VA 22901	DR *	VOGEL, MD, Scott Durand 920 East High Street Charlottesville VA 22901	OBG *	APPLIN, MD, Thomas Leo 5201 Carlton Street Bethesda, MD 20816	CHP
MARTIN, MD, Randolph P. 2560-B Ivy Road Charlottesville VA 22901	CD *	OOGHE, MD, Robt Barksdale 2125 Ivy Square, #6 Charlottesville VA 22903	IM	SELDEN, MD, Robt Francis 122 Overlook Dr Charlottesville VA 22901	PD	VOLKAN, MD, Vamk Djemal Uni Va Blue Ridge Hosp Charlottesville VA 22908	P	ARMSTRONG, MD, Charles L. 1451 Belle Haven Road Alexandria VA 22307	GP
MARTINEZ, MD, Horacio Duarte Route 6, Box 289B Charlottesville, VA 22901	AN *	ORR, MD, William A. Route 2, Box 2 Keswick, VA 22947	U	SHAFER JR., MD, Hubert Adams Box 170, Uni Va Med Ctr Charlottesville VA 22908	DR *	WALLENBORN, MD, White M 1000 E High St Charlottesville VA 22901	OTO *	AXELROD, MD, David Zuckerman 1451 Belle Haven Road Alexandria VA 22307	OBG *
MASLOFF, MD, James Irvin 1011 E Jefferson St Charlottesville VA 22901	U	OWEN JR., MD, John Atkinson Box 242, Uni Va Med Ctr Charlottesville VA 22908	DIA *	SHEPPE JR., MD, Wm Marco 2101 Arlington Blvd Charlottesville VA 22901	P *	WANEBO, MD, Harold J Box 181, Uni Va Med Ctr Charlottesville VA 22908	CRS *	BACAJ, MD, Taullah 810 Rapidan Court Alexandria VA 22304	OBG *
MASSARO, MD, Thomas A. Box 386, Uni Va Med Ctr Charlottesville VA 22908	PD	PAINE, MD, Wilmer H. 101 Stuart Place Charlottesville VA 22901	GP *	SHRUM, MD, Richard Coffman 400 Locust Ave Charlottesville VA 22901	GS *	WANG, MD, Gwo-Jaw Box 159, Uni Va Med Ctr Charlottesville VA 22908	ORS *	BAKER, MD, Wallace Edgar 6434 Brandon Ave Ste 1 Springfield Va 22150	GP
MC CUE III, MD, Frank C Box 243, Uni Va Med Ctr Charlottesville VA 22908	HS *	PEARL, MD, Elliott R. Box 386, Uni Va Med Ctr Charlottesville VA 22908	PD	SLOOP JR., MD, Frank B. 4300 Wellington Drive Charlottesville VA 22901	FOP	WEARY, MD, Peyton Edwin Box 134, Uni Va Med Ctr Charlottesville VA 22908	D *	BARNES, MD, Everett D. 1707 Osage St, #302 Alexandria, VA 22302	OBG *
MC GUIRE, MD, Lockhart Bemiss Box 158, Uni Va Med Ctr Charlottesville VA 22908	CD	PETLON II, MD, Ernest Wms 400 Locust Ave Charlottesville VA 22901	N	SMALLWOOD, MD, Harvey D 1011 E Jefferson St Charlottesville VA 22901	IM *	WEEKS, MD, Ruth B. 1224 W Main St, #501 Charlottesville VA 22903	P *	BAROT, MD, Lydia M 2616 Sherwood Hall Ln 404 Alexandria VA 22306	PD
MC LAUGHLIN, MD, Robert E Box 159, Uni Va Med Ctr Charlottesville VA 22908	ORS	PERRIELLO JR., MD, Vito A 1011 East Jefferson St Charlottesville VA 22901	PD	SMITH JR., MD, Arthur Morton 817 Fendall Terrace Charlottesville VA 22903	GYN *	WENZEL, MD, Richard Putnam Box 473, Uni Va Med Ctr Charlottesville VA 22908	ID	BAZO, MD, Albert James 1707 Osage Alexandria VA 22302	GP
MC LEAN, MD, Walter C 2700 Barracks Road Charlottesville VA 22901	OTO *	PHILLIPS II, MD, Lawrence H. 1856 Field Road Charlottesville VA 22903	N	SPAAR JR., MD, Albert P 2262 Ivy Drive Charlottesville VA 22903	P	WESTERVELT JR., MD, F. B. Box 133, Uni Va Med Ctr Charlottesville VA 22908	NEP	BEDNAR, MD, Stephen Jos 10654 Gunston Rd Lorton VA 22079	FP *
MCCORMACK, MD, Regina Claire 400 Locust Avenue Charlottesville VA 22901	IM	PHILLIPS, MD, Frank Harrison 932 E Jefferson Street Charlottesville VA 22901	FP	SPENCE, MD, Geo David 1005 E High St Charlottesville VA 22901	OPH *	WHEBY, MD, Munsey Stephen Box 502, Uni Va Med Ctr Charlottesville VA 22908	HEM *	BERGMAN, MD, Gary J. 5001 Seminary Rd Alexandria, VA 22311	PD
MCINTOCK, MD, M Gillian 11 Tennis Drive Charlottesville VA 22901	P	PIETSCH, MD, Richard Lake Mint Spring Valley Crozet VA 22932	OPH *	SPRADLIN, MD, W. W. 6 East, Blue Ridge Hosp Charlottesville VA 22901	P	WHITE, MD, Kerr Lachlan Route #1, Box 285 Stanardsville, VA 22973	IM	BERMAN, MD, Harold John 4921 Seminary Rd, #118-120 Alexandria VA 22311	D *
MENTZER JR., MD, Robert M. 205 Montvue Drive Charlottesville VA 22901	TS	POLLARD, MD, Wm Bryan 1149 Rose Hill Dr Charlottesville VA 22901	GP	STAMP, MD, Warren G Box 159, Uni Va Med Ctr Charlottesville VA 22908	ORS *	WHITEHILL, MD, Richard Box 159, Uni Va Med Ctr Charlottesville VA 22908	ORS *	BERNARD, MD, Peter G 2616 Sherwood Hall Ln Alexandria, VA 22306	N
MILLER JR., MD, Edward Doring Box 238, Uni Va Med Ctr Charlottesville VA 22908	AN	POPKIN, MD, Arnold B. 722 Rio Road West Charlottesville VA 22901	OPH	STEVENSON, MD, Ian Pretzman U Va Hosp Dept Psych Charlottesville VA 22908	P	WHITWORTH, MD, Claiborne G #1005 E High St Charlottesville VA 22901	OPH *	BERNANKE, MD, Abraham David 4660 Kenmore Ave #210 Alexandria VA 22304	IM
MILLER, MD, Charles W 912 E High Street Charlottesville VA 22901	ORS	POSADA, MD, Alejandro Emilio 2101 Arlington Blvd Charlottesville VA 22901	P	STONE, MD, James W Box 378, Uni Va Med Ctr Charlottesville VA 22908	IM *	WILHELM, MD, Morton C Box 181, Uni Va Med Ctr Charlottesville VA 22908	GS *	BHATIA, MD, Maya C 7111 Sussex Place Alexandria, VA 22307	CHP
MILLER, MD, James Quinter Box 394, Uni Va Med Ctr Charlottesville VA 22908	N *	POUTASSE, MD, Eugene F. Wintergreen Hm, Box 219 Nellysford, VA 22958	U	STRIDER, MD, David V 404 8th St N E Charlottesville VA 22901	ORS	WILKINS, MD, Paul Cole 1395 Hydraulic Road Charlottesville VA 22901	P	BIGELOW, MD, Llewellyn Barry 211 Lee Court Alexandria VA 22314	PD
MILLER, MD, Saml E Box 414, Uni Va Med Ctr Charlottesville VA 22908	FP *	PRINDLE, MD, Richard A 630 Kearsarge Circle Charlottesville VA 22901	PH	STURGLILL, MD, Benjamin Box 214, Uni Va Med Ctr Charlottesville VA 22908	PTH	WILLIAMS, MD, Gaylord Stone Box 376, Uni Va Med Ctr Charlottesville VA 22908	PS	BIGLEY JR., MD, Elmer C 2500 N Van Dorn St Alexandria VA 22302	ORS
MINOR, MD, Geo Ridgway Box 181, Uni Va Med Ctr Charlottesville VA 22908	TS	RABINOWITZ, MD, Seymour 307-C 2nd St, Nw Charlottesville VA 22901	P	SUSSMAN, MD, Michael David 2270 Ivy Road Charlottesville VA 22901	ORS *	WILLIAMSON, MD, Brian Richard Box 170, Uni Va Med Ctr Charlottesville VA 22908	NM *	BLACKMON, MD, Wayne D. 4501 Connecticut Ave, #104 Washington, D.C 20008	P
MOGA, MD, David B 820 East High Street Charlottesville VA 22901	ORS	REID, MD, Robt Allen Box 432, Uni Va Med Ctr Charlottesville VA 22908	IM	SUTPHEN, MD, James L. Box 386, Uni Va Med Ctr Charlottesville VA 22908	PD	WILSON JR., MD, Lester A Box 387, Uni Va Med Ctr Charlottesville VA 22908	OBG *	BLAKE, MD, Jeffrey D. 5001 Seminary Rd, #117 Alexandria, VA 22311	PD *
MOHLER JR., MD, Danl N Box 395, Uni Va Med Ctr Charlottesville VA 22908	HEM *	RESPESS, MD, James C Box 145, Uni Va Med Ctr Charlottesville VA 22908	GE *	SYDOR JR., MD, Thos Austin 1000 E High St Charlottesville VA 22901	OTO *	WILSON, MD, Edward Croft Box 145, Uni Va Med Ctr Charlottesville VA 22908	GE	BLAKEY, MD, Hubert H. 1203 N. Ouaker Lane Alexandria, VA 22032	P
MONTGOMERY, MD, Howard Arthur 1101 East High Street Charlottesville VA 22901	OBG *	RICHARDSON, MD, Donald R. 1000 East High Street Charlottesville VA 22901	D	SYDOR, MD, Robt W. 2019 Tate Springs Rd Lynchburg, VA 24501	ORS	WILSON, MD, William G. Box 386, Uni Va Med Ctr Charlottesville VA 22908	PD	BOGER, MD, David S. 1601 18th Street, Nw Washington, DC 20009	P
MOODY, MD, David T. 1602 Gordon Ave Charlottesville VA 22903	P *	RIDDERVOLD, MD, Hans Olav Box 170, Uni Va Med Ctr Charlottesville VA 22908	DR *	TALBOTT, MD, William G. 219 Montvue Drive Charlottesville VA 22901	EM	WOOD, MD, James Burnley 1646 Keith Valley Rd Charlottesville VA 22901	PD	BONDAREFF, MD, Erwin Allen 4201 Ormond Ave Alexandria VA 22304	PD
MOODY, MD, Wm Edward Box 217 Scottsville VA 24590	GP *	RIPBERGER, MD, Frank M. Box 378, Uni Va Med Ctr Charlottesville VA 22908	PD	TATAR, MD, Steven Andrew 1011 E Jefferson St Charlottesville VA 22901	IM	WORD, MD, Benj Harrison 1101 East High Street Charlottesville VA 22901	OBG *	BRAYSHAW, MD, James Rodney 4660 Kenmore Ave #210 Alexandria VA 22304	IM

\* = AMA membership



BREGMAN, MD, Robt L P O Box 9620 Alexandria VA 22304	PD	DESOUZA, MD, Romaldo F X 4801 Kenmore Ave, # 102 Alexandria VA 22304	IM	GRECO, MD, Philip Scot 5249 Duke Street, #307 Alexandria VA 22304	P	KHACHIKIAN, MD, Grigor 5249 Duke Street Alexandria, VA 22304	OBG	MOON, MD, Young Ho 1451 Belle Haven Rd Alexandria VA 22307	CD
BRIGUGLIO, MD, Philip 5791 Winston Ct, #160 Alexandria VA 22311	OBG	DEUTSCH, MD, Alan Seth 5001 Seminary Rd Alexandria VA 22311	GS	GREEN, MD, Ira Joel 4660 Kenmore Ave, #608 Alexandria VA 22304	DR	KHOMAMI-RAMSEY, MD, Ali 8996 Fern Park Drive Burke, VA 22015	OBG	MOON, MD, Young Sun 7400 Park Terrace Dr Alexandria VA 22307	
BROWN, MD, James Geo 1707 Osage St Alexandria VA 22302	GS	DI PINTO, MD, Felix R. 6300 Stevenson Ave, #B Alexandria, VA 22304	IM	GREENSPAN, MD, Robert Edward 5249 Duke St Suite 409 Alexandria VA 22304	NEP	KLOUSIA, MD, John Walter 3323 Duke Street Alexandria VA 22314	U	MOORE III, MD, James Merton 2616 Sherwood Hall Ln, #303 Alexandria VA 22306	GS
BRUNO JR., MD, John A 1300 Lafayette Drive Alexandria VA 22308	ORS	DLUHY, MD, John Michael 3709 Ingomar Street, Nw Washington, D.C. 20015	P	GRETHER, MD, Eugene Rudol 5808 Cannon Lane Alexandria VA 22303	GP	KOLANSKY, MD, Saul Kalman 1100 Collingwood Rd Alexandria VA 22308	CHP	MOSCHELLA III, MD, Ralph 7116 Park Terrence Dr Alexandria, VA 22307	AN
BUENAVENTURA, MD, Francisco A 9211 Volunteer Dr Alexandria VA 22309	AN	DOBRYNSKI, MD, Robt F 5226 Dawes Ave Bldg D Alexandria VA 22311	HEM	GUTHRIE, MD, Norman David 101 S Whiting St #212 Alexandria VA 22304	P	KORKOSZ, MD, Tanya J. 614 S. Columbus Street Alexandria, VA 22314	P	MOSS, MD, James Mercer 1707 Osage St Alexandria VA 22302	DIA
BUHAIN, MD, Willfrido J 6300 Stevenson Ave, B Alexandria VA 22304	PUD	ELSBERG III, MD, Paul 914 Timberbranch Pkwy Alexandria VA 22302	AN	HAGER, MD, Ewald Joseph 2616 Sherwood Hall Ln, #408 Alexandria VA 22302	OTO	KOSLOW, MD, Joel Lester 4921 Seminary Rd Apt 115 Alexandria VA 22311	IM	MOUROT, MD, Arthur J 914 Prince St, #3 Alexandria VA 22314	GS
BUTLER JR., MD, Bruce 5021 Seminary Rd Alexandria VA 22311	HS	ENGH, MD, Charles A 2465 Army-Navy Dr Arlington VA 22206	ORS	HAKIM, MD, Arcadius H. 1716 Braddock Road Alexandria VA 22302	OTO	KUYKENDALL, MD, Harry Canter 4921 Seminary Rd Alexandria VA 22311	FP	MUNTERS, MD, Manfreds 1300 Lafayette Drive Alexandria, VA 22308	ORS
BUTLER, MD, Lilia G 3823 Duke St Alexandria VA 22304	PD	ERAGAN, MD, Mehmet Aril 5001 Seminary Road Alexandria VA 22311	A	HALTER, MD, Paul Edmund 5807 Ashfield Road Alexandria VA 22310	OBG	LARSEN JR., MD, Kenneth T Box 7 Route One Marshall VA 22115	EM	NACHAJSKI, MD, Peter John P O Box 9620 Alexandria VA 22304	PD
CALDWELL, MD, John Leo 2030 16th Street Washington DC 20009	DR	ERICKSEN, MD, Thomas W. 4660 Kenmore Alexandria, VA 22304	IM	HAMMACK, MD, Phillip Larry 5274 Dawes Avenue Alexandria VA 22311	GS	LEABHART JR., MD, John W 2500 N Van Dorn St Alexandria VA 22302	ORS	NADLER, MD, Joel Bruce 5249 Duke St, #309 Alexandria VA 22304	IM
CARDONA, MD, Angel A 517 N St Asaph St Alexandria VA 22314	PH	ERIM, MD, Zeki 2616 Sherwood Hall Lane Alexandria VA 22306	R	HAUT, MD, Donald David 312 S. Washington St. 2D Alexandria, VA 22314	IM	LEE, MD, Chong Wook 4901 Seminary Rd #108 Alexandria VA 22311	TS	NALLS, MD, Walter L Inverwood Boyce VA 22620	IM
CARROLL JR., MD, Frank A 1707 Osage St Alexandria VA 22302	FP	FERRELL JR., MD, Haskins 312 S Washington St Alexandria VA 22314	OBG	HECKER, MD, Carlos Metsch 3450 N. Beauregard Alexandria, VA 22302	P	LEE, MD, Margaret C 622 W Braddock Road Alexandria VA 22302	AN	NIGRO JR., MD, Michael F 5001 Seminary Rd, #225 Alexandria VA 22311	GS
CATALDO, MD, Jos Richard 2616 Sherwood Hall Ln, #207 Alexandria VA 22306	FP	FIFER, MD, Carson Lee 1100 St Stephens Rd Alexandria VA 22304	GYN	HERNANDEZ, MD, Antonio 5249 Duke Street Alexandria, VA 22304	OBG	LEMESHEWSKY, MD, Geo P 312 S. Washington St. 6A Alexandria VA 22314	GP	NIGRO, MD, Bernard Angelo 700 Duke Street Alexandria VA 22314	P
CAVENDER, MD, Wm Francis 7711 Lookout Court Alexandria VA 22306	P	FLIS JR., MD, Jos F 8606 Sandy Ridge Ct Fairfax VA 22031	GE	HERON JR., MD, A. Roy 732 North Washington St Alexandria VA 22314	FP	LEVIN, MD, Stephen M 5021 Seminary Rd Alexandria VA 22311	ORS	NOER, MD, H ROLL 2465 Army-Navy Dr Arlington VA 22206	ORS
CHAO, MD, Yu-Hua 2005 Mason Hill Drive Alexandria VA 22307	IM	FRANCIS JR., MD, Cleveland 8101 Hinson Farm Road Alexandria VA 22306	CD	HERR JR., MD, Austin Alexis 4660 Kenmore Ave Alexandria VA 22304	OTO	LIEBERMAN, MD, Michael David 1302 Lafayette Drive Alexandria VA 22308	FP	O'CONNOR, MD, John J 1123 St Stephens Rd Alexandria, VA 22304	AN
CHAPMAN, MD, A Bradley 2059 Huntington Ave Alexandria VA 22303	CHP	FRIED, MD, William A 4708 Kenmore Ave Alexandria, VA 22304	PD	HERSH, MD, Stephen Robt 2500 North Van Dorn St Alexandria VA 22302	DR	LITTLEFIELD, MD, Jerald J. 422 S. Washington St Alexandria, VA 22314	OPH	OCEAN, MD, Ronald Hugh 2616 Sherwood Hall Lane Alexandria VA 22306	GS
CHAPMAN, MD, Val Loren 9111 Meadow Rue Lane Annandale, VA 22003	FP	FRIEDLIS, MD, Mayo 5201 Leesburg Pike, #102 Falls Church, VA 22041	PM	HERTZBERG, MD, Michael 303 Cameron Street Alexandria VA 22314	P	LONG, MD, Albert Emanuel 3012 Peacock Lane Tampa, FL 33618	OPH	ORDONEZ, MD, Mario Andres 2218 Sherwood Hall Lane Alexandria VA 22306	P
CHARNEY, MD, David L 414 N Union St Alexandria VA 22314	P	FRIEDMAN, MD, Michael Herbert 1707 Osage Street Alexandria VA 22302	DR	HINDLE JR., MD, Wm Vincent Alexandria Hospital Alexandria VA 22314	DR	LONGO, MD, Antonio Miguel 4801 Kenmore Ave Alexandria VA 22304	PUD	OSHEROFF, MD, Raphael J 5249 Duke St, #203 Alexandria VA 22304	NEP
CHEN, MD, Cheng-Nan 312 S. Washington St, #2-B Alexandria VA 22314	US	GAHRES, MD, Edward E 5021 Seminary Rd Alexandria VA 22311	OBG	HODGES JR., MD, Emory Falcon 312 South Washington Street Alexandria VA 22314	P	LOPEZ-TOCA, MD, Ruben 6301 Stevenson Ave Apt 417 Alexandria VA 22304	IM	OXENHANDLER, MD, Donald C 2807 Duke Street Alexandria VA 22314	N
CHOI, MD, Walter Sik 7215 Burtonwood Dr Alexandria VA 22307	R	GALLER, MD, Floyd Bruce 1616 18th Street, N.W. Washington, D.C. 20009	P	HOWE, MD, James Robt 2807 Duke Street Alexandria VA 22314	NS	LOUGHRIDGE, MD, Chalmers A 3901 Rive Dr Alexandria VA 22309	EM	PALMER, MD, Richard E P O Box 1229 Alexandria VA 22313	PTH
CHUNG, MD, Kyung Yil 5008 Heritage Lane Alexandria, VA 22311	AN	GALLINI, MD, Marc Robert 8228 Mount Vernon Highway Alexandria VA 22309	FP	HOYLE, MD, John D 312 S Washington St Alexandria VA 22314	GS	LOWEN, MD, Beal Aptheke 8101 Hinson Farm Road Alexandria VA 22306	IM	PALUCH III, MD, Simon 1610 Brandon Ave Springfield VA 22150	IM
CIGTAY, MD, Atilla Sakir 312 S Washington St Alexandria VA 22314	DR	GAUGHAN, MD, Robert T 2805 Duke Street Alexandria VA 22314	ORS	HUBERMAN, MD, Richard 4660 Kenmore Ave Alexandria VA 22304	OPH	LUCCIOLI JR., MD, Lucio 4600 King St, #5-E Alexandria, VA 22302	OTO	PARKINSON, MD, Dee R 4320 Seminary Rd Alexandria VA 22314	PTH
CLARK, MD, Laurence J 1451 Belle Haven Road Alexandria, VA 22307		GIAMMITTORIO, MD, David C. 5249 Duke St, #308 Alexandria, VA 22304	OBG	HUDSON, MD, Chas A 501 Crown View Dr Alexandria VA 22314	CD	LUKOWSKY, MD, Gerhard Hans 312 S Washington St Alexandria VA 22314	IM	PARVIN III, MD, Shahindokht 4660 Kenmore Ave #408 Alexandria VA 22304	P
COCHRAN, MD, John Wesley 2807 Duke Street Alexandria, VA 22314	IM	GIANNUZZI, MD, Vito A. 1451 Belle Haven Road Alexandria, VA 22307	PD	HUFF JR., MD, Wm Thos 810 Green Street Alexandria, VA 22314	AN	MAHINPOUR, MD, Siavash 6439 Lakeview Drive Falls Church, VA 22041	ORS	PAULSON, MD, John D 4801 Kenmore Ave Alexandria VA 22304	OBG
COLLIS, MD, Peter B 4506 49th Street Nw Washington DC 20016	EM	GIBSON, MD, John Eugene 5249 Duke St Ste 5 Alexandria VA 22304	D	HURTADO, MD, Rodrigo C. 3450 North Beauregard Alexandria, VA 22302	AI	MANN, MD, James Packard 3709 S George Mason, #1505 Falls Church, VA 22041	PUD	PEPPER, MD, Franklin Jay 4600 Duke St Ste 424 Alexandria VA 22304	P
CONLEY, MD, Eugene Jos 700 Duke St Alexandria VA 22314	P	GIBSON, MD, Robert John 8101 Hinson Farm Rd, #118 Alexandria, VA 22306	FP	IRANYI, MD, Magdolna A 3541 W Braddock Rd Alexandria VA 22302	PD	MARAK JR., MD, Geo Edward 2059 Huntington Ave, P-14 Alexandria VA 22303	OPH	PEREIRA, MD, Isabel T 6301 Stevenson Ave Apt 417 Alexandria VA 22304	P
CROSS, MD, Phyllis De Carlo 6304 Buffalo Ridge Road Falls Church, VA 22044	GP	GILLIGAN JR., MD, John Henry 1707 Osage St Alexandria VA 22302	OPH	JAMMES, MD, Juan Luis 6421 Waterway Drive Falls Church VA 22044	N	MAY, MD, Dean Francis 2004 White Oak Dr Alexandria VA 22306	PD	PETERS, MD, Lawrence S. 4660 Kenmore Ave., S-202 Alexandria, VA 22304	GE
DATOC, MD, Roberto L 4701 Kenmore Ave, #119 Alexandria VA 22304	US	GILLINSON, MD, Roy Stuart 4901 Seminary Rd, #104 Alexandria VA 22311	AN	JENKINS, MD, Chas E 607 Ramsey St Alexandria VA 22301	GP	MC DADE, MD, John Patrick 4320 Seminary Rd Alexandria VA 22314	EM	PICOT, MD, Harrison 5249 Duke St 408 Alexandria VA 22304	OBG
DAVALOS, MD, Hugo A 2616 Sherwood Hall Lane Alexandria VA 22306	US	GILMORE, MD, Bruce Leslie 5021 Seminary Rd Alexandria VA 22311	IM	JOHNSON, MD, Kenneth Roger 1700 N Van Dorn St Alexandria VA 22304	OBG	MC GOUGH, MD, Thos F 3112 Holly St Alexandria VA 22305	FP	PREUSS, MD, James Wm 2807 Duke Street Alexandria VA 22314	NS
DAVID-NELSON, MD, Margit A 8102 Ashtonbirch Dr Springfield VA 22152	PTH	GOLDBERGER, MD, Stephen G. 7801 Old Branch Avenue Clinton, MD 20735	NEP	JONES JR., MD, Benj C 312 S Washington St Alexandria VA 22314	CD	MC KNELLY, MD, Larry Oren 1707 Osage St Suite 400 Alexandria VA 22302	PS	PRICE, MD, Kazuko Kukita 4701 Kenmore Ave Alexandria VA 22304	OBG
DAVIDSON, MD, Stuart L 1300 Lafayette Drive Alexandria, VA 22308	ORS	GOLDBLATT, MD, Seymour Zonald 5001 Seminary Rd Alexandria VA 22311	PD	JOSE, MD, Nora D 2616 Sherwood Hall Ln, #404 Alexandria VA 22306	PD	MELONI, MD, Chas Robt 1707 Osage St Alexandria VA 22302	DIA	PRICE, MD, Richard A. 1800 Hunting Cove Place Alexandria, VA 22307	EM
DAVIES, MD, John Benj 5212 Dawes Avenue Alexandria VA 22311	P	GOLDHAMMER, MD, Leo 611 S Carlin Sprgs Rd, #303 Arlington, VA 22204	N	JOSE, MD, Pedro A 2616 Sherwood Hall Ln, #404 Alexandria VA 22306	OS	MICHENER, MD, Frank Ervine 9400 Ferry Landing Ct Alexandria VA 22309	CHP	PRUGH, MD, Merrill Frederick 4600 King Street #2-L Alexandria VA 22302	GE
DAVIS, MD, Donald Irvin 7805 Elba Rd Alexandria VA 22306	P	GONDOR, MD, Leslie Paul 3541 W Braddock Rd Alexandria VA 22302	US	KAMEL, MD, Medhat Mohamed 4810 Beauregard St, #304 Alexandria VA 22312	GS	MILLER, MD, Geo Francis 1707 Osage St Alexandria VA 22302	OTO	PUGH, MD, H Lamont 6251 Old Dominion Dr Mc Lean VA 22101	GS
DE VOCHT, MD, Ludovic Jules 4421 Seminary Rd Alexandria VA 22304	OBG	GONZALEZ, MD, Miguel H 5242 Dawes Ave, #3-H Alexandria VA 22311	CD	KAPLIN, MD, Arnold Jay 5021 Seminary Rd Alexandria VA 22311	P	MILLER, MD, Irvin S 5021 Fort Hill Dr Alexandria, VA 22303	OBX	RAJAE III, MD, Shahabeddin Alexandria VA 22310	OBG
DELANEY JR., MD, Martin D 329 N Washington St Alexandria VA 22314	US	GOODENBERGER, MD, D. M. 3718 Woodley Drive Alexandria VA 22309	EM	KARPICK, MD, Ronald John 3413 Rusticway Lane Falls Church VA 22044	PUD	MILLS JR., MD, James D 1304 Warrington Place Alexandria VA 22307	EM	REDDING, MD, Gorman Jos 2616 Sherwood Hall Ln, #407 Alexandria VA 22306	GP
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DELORME, MD, Donald P 2616 Sherwood Hall La 402 Alexandria VA 22306	GYN	GRAND, MD, Bernard 5216 Dawes Ave Alexandria VA 22311	PUD	KENDRICK, MD, Marvin H. 720 W Braddock Road Alexandria, VA 22302	GP	MOLCHON, MD, Andrew B. 5001 Seminary Rd, #106 Alexandria, VA 22311	GP	REID, MD, Wm Mitchell 5001 Seminary Rd Alexandria VA 22311	GS

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RHAME, MD, Richard Coleman 1707 Osage St Alexandria VA 22302	U *	SMIRNOTOPOULOS, MD, Thomas Route 1, Box 251 Charles Town, WV 24514	IM	WERTHEIM, MD, Raymond B 220 S. Washington St Alexandria, VA 22314	CHP	NUNLEY, MD, Wallace Clay P O Box 76 Clifton Forge VA 24422	US *	BERRY, MD, H Lee 1414 Laburnum St McLean VA 22101	GS *
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ROBERTS, MD, John Edmund 312 S Washington St Alexandria VA 22314	GP *	SPELLER, MD, Jeffrey L 805 Franklin Street Alexandria, VA 22314	P	WILMOT, MD, Benneville Dayton 904 Danton Lane Alexandria VA 22308	PD *	SANKAR, MD, Krishna Mallow Road Covington VA 24426	OPH *	BOLAND, MD, Brian J 1715 N Geo Mason Dr, #406 Arlington, VA 22205	IM *
ROBSON, MD, Scott M 128 South Royal St Alexandria, VA 22314	GP	STEIN JR., MD, Jerome 1451 Belle Haven Rd Alexandria VA 22307	OBG *	WINELAND, MD, Robt K 2213 Marthas Rd Alexandria, VA 22307	PD *	SCHROEDER, MD, Mark D. Box 547 Clifton Forge, VA 24422	IM *	BORGES, MD, Philip M. 6349 Crosswoods Drive Falls Church, VA 22044	U *
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ROMANSKY, MD, Stephen Hess 6526 Jay Miller Drive Falls Church, VA 22041	P	STONE, MD, Wm M 8492 Richmond Hwy Alexandria VA 22309	CD	WOLFF JR., MD, Herbert D 1600 Westbrook Ave, Apt 325 Richmond, VA 23227	U *	SNYDER, MD, James L Box 531, Emmet Med Surg Clifton Forge VA 24422	U *	BRISCOE, MD, Wm Cole 1812 Baldwin Drive McLean, VA 22101	CHP *
ROSENFELD, MD, Stephen P 4660 Kenmore Ave, #410 Alexandria VA 22304	CD *	SULLIVAN, MD, Thos Jos 4921 Seminary Rd Alexandria VA 22311	PD	WOODSIDE, MD, Jack R 9125 Christopher St Fairfax VA 22031	AN *	VERMILYA, MD, Walter E P O Box 46 Clifton Forge VA 24422	GP	BROWN, MD, Thos Mc Pherson 2465 Army-Navy Drive Arlington VA 22206	RHU *
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RUONA, MD, Luanne 1198 Janney's Lane Alexandria, VA 22302	P	SWAN, MD, Harvey Frank P O Box 34014 Bethesda, MD 20817	D	ZALCMAN, MD, Steven Jay 2100 Whiteoaks Dr Alexandria VA 22306	P	WINN, MD, Thos Meredith 108 N Marion Ave Covington VA 24426	OPH *	BUCUR, MD, John C 7 Corners Med Bldg Falls Church VA 22044	NS *
RYAN, MD, Richard Herrick 4921 Seminary Rd Alexandria VA 22311	PD	SWISHER, MD, Forrest M 2500 N Ven Dorn St Alexandria VA 22302	ORS	ZEAVIN, MD, Bernard H 5055 Seminary Rd Ste 111 Alexandria VA 22311	OPH *	WYSOR, MD, Frank Laird P O Box 146 Clifton Forge VA 24422	OPH *	BULLOCK, MD, Robt Graham 611 S Carlyn Sprs Rd, #306 Arlington VA 22204	IM *
RYU, MD, Jai Yol 2616 Sherwood Hall Ln, #301 Alexandria VA 22302	FP *	SYME, MD, Robert H 4660 Kenmore Ave, #419 Alexandria VA 22304	OBG *	ZEHFUSS, MD, Paul E 1203 N Ouaker Lane Alexandria VA 22302	OPH *	AMHERST-NELSON			
SALCEDO, MD, Hernando P 1707 Osage St Alexandria VA 22302	U *	TACKTILL, MD, Norman 1451 Belle Haven Road Alexandria, VA 22307	OBG *	ALLEGHANY-BATH				GAMBLE, MD, James H P O Box 217 Lovington VA 22949	GP *
SANDLER, MD, Allen H 1704 37th Street, Nw Washington, DC 20007	P	TALBOT, MD, Frank James 2616 Sherwood Hall Lane Alexandria VA 22306	CD	ADRALES, MD, Mamerto 322 W Riverside Street Covington, VA 24426	GS *	RICH, MD, Elizabeth J Pedlar Mills Monroe, VA 24574	N	BUTLER, MD, Thomas Parke 1715 N Geo Mason Dr, #307 Arlington VA 22205	IM *
SANNER, MD, M Oue 5927 Cauba Ct Alexandria VA 22310	GP	TAMARIZ, MD, Theodore E 312 S Washington St Alexandria VA 22314	US *	ALLEN, MD, Edward G P O Box 123 Clifton Forge VA 24422	R *	ARLINGTON			
SAPPINGTON JR., MD, Richard F 4600 King St, #4-E Alexandria, VA 22302	PUD *	TEMUCIN, MD, Oguz P O Box 9203 Alexandria VA 22304	AN *	BALLOU, MD, Chas F Laurel Hill Clifton Forge VA 24422	IM *	ALEXANDER, MD, John Emanuel 611 S Carlyn Springs Road Arlington, VA 22204	PS	CAMBAREH, MD, Richard J. 1715 N George Mason Dr Arlington, VA 22205	ON *
SAWMILLER, MD, Samuel 2500 N Van Dorn St Alexandria VA 22302	ORS	THOMPSON, MD, Harry Glenn 5249 Duke St, #107 Alexandria VA 22304	OBG *	CHUCKER, MD, Geo N P O Box 608 Clifton Forge VA 24422	R *	ALEXANDER, MD, Michael B. 611 S. Carlyn Springs Road Suite 203 Arlington, VA 22204	IM	CARDENAS III, MD, Francisco Arlington Hosp Arlington VA 22205	GS *
SCHIFFMAN, MD, Joel H 2805 Duke Street Alexandria VA 22314	ORS *	TINKER, MD, Bruce P. 8101 Hinson Farm Rd, #114 Alexandria VA 22306	IM *	CLATERBAUGH, MD, Raymond L 1130 Grace Ave Clifton Forge VA 24422	GP	ALFONSO, MD, Raymundo P 4600 King Court, #4-R Alexandria, VA 22302	GP	CASEY, MD, Catherine Sue 1715 N George Mason Dr, #205 Arlington VA 22205	PD
SCHKOLNIK JR., MD, Ronald 6613 Goldsboro Rd Falls Church VA 22042	AN	TITUS JR., MD, Jonathan 8228 Mt Vernon Highway Alexandria, VA 22309	FP	DAKERMANDJI, MD, Farid P O Box 111 Clifton Forge, VA 24422	AN	AMEDEO, MD, Ralph M 9500 Ocala Street Silver Spring, MD 20901	IM	CHADAB, MD, Marvin 6192 Oxon Hill Road Oxon Hill MD 20021	D
SCHWARTZ, MD, Harvey Albert 7716 Delafeld Place Alexandria VA 22306	IM	TITUS, MD, Frederick Preston 738 Fontaine St Alexandria VA 22302	OBG	DENIUS, MD, Larry Richard Virginia Bldg Hot Springs VA 24445	FP	ANDERSON, MD, Edward Lee 1310 N Courthouse Rd Arlington, VA 22201	IM *	CHALMETA, MD, Alberto 611 S Carlyn Spgs Rd, #208 Arlington VA 22204	N
SCHWARTZ, MD, James R. 2616 Sherwood Hall Lane Alexandria, VA 22306	ORS	TODHUNTER, MD, Richard Boyd 8316 Arlington Blvd, #502 Fairfax, VA 22031	PD *	EDMONDS JR., MD, Meade C Box 531 Clifton Forge VA 24422	GS *	APTER, MD, Ronald Alan 2001 Columbia Pike Arlington VA 22204	CD *	CHAPMAN, MD, Dorothea 5501 Seminary Rd, #1405 Falls Church, VA 22041	IM
SCOTT, MD, Pierre Brutsche 3541 W Braddock Rd Alexandria VA 22302	OPH *	TOKARZ, MD, John Pat 1707 Osage St, #402 Alexandria, VA 22302	FP *	EDMONDS, MD, Julia Emmett P O Box 108 Clifton Forge VA 24422	PD	ARAKAKY-SALAZAR, MD, Abel 9722 Biggers Road Burke, VA 22015	IM *	CIOFALO, MD, Carol Ellen 1009 N George Meson Dr Arlington, VA 22205	OBG
SEMMES, MD, Benedict J. 408 Yuma Street, Nw Washington, DC 20016	IM *	TRIMBER, MD, Connell James Box 355 Alexandria VA 22313	OPH	ELLIS, MD, Wm Jos 425 W Locust St Covington VA 24426	GP	ASCUNCE, MD, Gil 1715 N George Mason Dr, #410 Arlington, VA 22205	IM	COLE, MD, Ralph J 4301 Columbie Pike Arlington VA 22204	OBG
SENDI, MD, Houchang 5425 Duke Street Alexandria VA 22304	PS	TUAZON, MD, Oscar C 5249 Duke St Ste 205 Alexandria VA 22304	GS *	FINESTONE, MD, Alvin Wm P O Box 608 Clifton Forge VA 24422	R *	AUSTIN, MD, Harvey W 1776 Old Meadow Road McLean, VA 22102	PS *	CONTIS, MD, George Peter 1716 Wilson Blvd Arlington, VA 22209	PH *
SHAFFER, MD, Stephen R 3701 George Mason Drive Apt C-2-N Falls Church, VA 22041	HS	UBELHART, MD, Charles Robert 2805 Duke Street Alexandria, VA 22314	ORS *	FISCHER, MD, Geo L P O Drawer 547 Clifton Forge VA 24422	IM *	EVERY, MD, Gordon Lee 3801 N Fairfax Dr, #70 Arlington, VA 22203	ORS *	CORNELL, MD, Roger Detlet 6500 Clifton Rd Clifton VA 22024	R
SHANKMAN JR., MD, Sidney 4720 Montgomery Lane Bethesda MD 20814	CHP *	UENO, MD, Winston Mizuo 5226 Dawes Ave Alexandria VA 22311	IM *	GOINGS, MD, Ronald Steven 200 Church Street Clifton Forge, VA 24422	FP	BACARRA, MD, Abraham V 9709 Singleton Drive Bethesda, MD 20817	AN	COSTESCU, MD, Sanda 3801 N Fairfax, #20 Arlington, VA 22203	IM *
SHAUER, MD, Alan B. 2616 Sherwood Hall Ln, #209 Alexandria, VA 22306	GE	UPTON, MD, David Leslie 208 N Pitt St Alexandria VA 22314	P *	HALL, MD, Aubrey Carlyle P O Drawer 547 Clifton Forge VA 24422	IM *	BACKER, MD, Jos Anthony 6962 Kyleakin Ct McLean VA 22101	DR	COTTS, MD, Gerhard K 333 S Glebe Rd #410 Arlington VA 22204	P *
SHEELY, MD, Wm Edward 1231 Kingston Ave Alexandria VA 22302	R *	VARGAS-MERA, MD, Filiberto 8808 Anne Tucker Lane Alexandria VA 22309	IM *	HAMILTON, MD, Thomas F. 315 West Main Street Covington, VA 24426	FP	BAGLEY, MD, Clifford E 12605 Hazelwood Dr Nokesville VA 22123	U *	COUDON, MD, Wilson L. 1400 South Joyce St Apt A-102 Arlington, VA 22202	PUD
SHIH, MD, Teh-Chang 3705 Templeton Place Alexandria VA 22304	IM *	WARSHAUER JR., MD, Sanford 1451 Belle Haven Rd Alexandria VA 22307	OBG *	HARNBERGER, MD, James Power Box P Hot Springs VA 24445	OM *	BAJWA, MD, Manjit Rajinder 1007 Heather Hill Ct McLean, VA 22101	AN	CROSSLAND, MD, Stanley G. 3801 N Fairfax Dr, #503, S Arlington, VA 22203	GS
SHUMAM, MD, Lawrence Henry 4810 Beauregard St Ste 303 Alexandria VA 22312	OBG	WATSON, MD, John C 808 Prince St Alexandria VA 22314	CD	JOHNSON JR., MD, Walter W 202 Walnut St Covington VA 24426	GP *	BARTRAM, MD, Scott F. 200 Little Falls St, #301 Falls Church, VA 22046	GP	CSATARY, MD, Laszlo K 1600 South Eads Street Arlington, VA 22202	GP *
SILBERMAN, MD, Edward K. 2304 N Jackson Street Arlington, VA 22201	P	WEAVER JR., MD, Wm Jack Box 759 Locust Grove, VA 22508	EM *	LE HEW, MD, Allen Edwin C & O Hospital Clifton Forge VA 24472	IM *	BASTIEN, MD, Henry L 3801 Fairfax Dr Arlington VA 22203	OPH *	CURRY, MD, John Lamar 6231 Leesburg Pike, #612 Falls Church, VA 22044	GP
SIMS, MD, John Adrian 2729 King St Alexandria VA 22302	GER *	WEINBERGER, MD, Daniel R St Elizabeths Hospital, #536 Washington DC 20032	P	LUKE, MD, Mary Jane Box 351 - Glencairn Covington, VA 24426	PDC	BAZAZ, MD, Lokesh K. 611 S Carlyn Spgs Rd, #408 Arlington VA 22204	N	DALEY, MD, Timothy Horton 1715 N George Mason Dr, #205 Arlington VA 22205	PD
SLATE, MD, Herman Ivan Box 1142 815 Prince St Alexandria VA 22313	OS *	WEINSHANK, MD, Herbert S 10720 Main St Fairfax VA 22030	PYA	MINEIRO, MD, Luiz E G Box 531, Emmet Med Surg Clifton Forge VA 24422	GS *	BEINSTEIN, MD, Joseph 611 S Carlyn Springs Rd, #302 Arlington VA 22204	CD *	DALTON, MD, Henry Tucker 3801 N Fairfax Dr, #105 Arlington, VA 22203	OPH *

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DAVITT, MD, Mary Catherine 1701 North George Mason Dr Arlington VA 22205	PD	HOAR, MD, Barbara R. 1021 Prince St, #100 Alexandria, VA 22314	CHP	LINN, MD, James John 7277 Evans Mill Road Mc Lean, VA 22101	PTH	MYERS, MD, Lynne Beth Davis 107 N Virginia Falls Church VA 22046	PD	ROSEBLAT, MD, Aldo M. 6316 Castle Pl, #2E Falls Church, VA 22044	NS
DAVOUDLARIAN, MD, David K 5205 Leesburg Pike Falls Church VA 22041	OBG	HOARE, MD, Raymond Robert 1715 Geo Mason Dr, #107 Arlington VA 22205	CD	LISZKA, MD, Victor L. 3543 West Braddock Road Alexandria, VA 22302	GS	NACCASH, MD, Edmund P 3321 Sydenham St Fairfax VA 22031	GYN	ROSENBAUM JR, MD, Meyer 939 S Wakefield St Arlington VA 22204	OBG
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DIETZ JR, MD, Richard F. 2517 North Glebe Rd Arlington VA 22207	IM	HONG, MD, Seung Kook 9003 Streamview Lane Vienna, VA 22180	AN	LONG, MD, George E. 9947 Longford Court Vienna, VA 22180	GP	NEEFE, MD, Dana Lynne 1516 Hardwood Lane Mc Lean VA 22101	IM	ROTCHFORD, MD, James Patrick 3801 N Fairfax Dr Suite 52 Arlington VA 22203	D
DODSON, MD, Wm Walter 4600 Conn Ave Nw, #111 Washington, D.C 20008	P	HOPWOOD, MD, Herbert G 1715 N George Mason Dr Arlington VA 22205	OBG	LOPEZ, MD, Rodolfo L. 2210 Fort Ward Place Alexandria, VA 22304	GS	NEU, MD, Robt Bernard 6141 Lee Highway Arlington VA 22205	P	RUCKSTUHL, MD, Lily 7545 Idylwood Rd Falls Church VA 22043	IM
DOLAN JR, MD, Wm David 1701 N George Mason Dr Arlington VA 22205	PTH	HOWARD, MD, Robt L 2745 N. Radford St Arlington VA 22207	CD	LUMPKIN, MD, Martha Ray 611 S Carlin Sprgs Rd, #103 Arlington, VA 22204	IM	NGO, MD, Thuan Dinh 3801 N Fairfax Dr, #201 Arlington, VA 22203	GP	RYAN, MD, John Edward 3801 N Fairfax Dr Arlington VA 22203	OBG
DOUGHERTY, MD, William E. 1715 N Geo Mason Dr, #404 Arlington VA 22205	U	HOYME, MD, Jane C. 2640 North Ohio St Arlington VA 22207	IM	LUNDEEN, MD, Wm Bruce Arlington Hospital Arlington VA 22205	TR	NIRSCHL, MD, Robert P 1715 N George Mason Dr Arlington VA 22205	ORS	RYAN, MD, Robert Francis 1715 N George Mason Dr, #304 Arlington, VA 22205	IM
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ELLIOTT, MD, John J. 3801 N Fairfax Drive Arlington VA 22201	OBG	IMBURG, MD, Jerome 1911 Windmill Lane Alexandria VA 22307	PD	MACIULLA, MD, Louis J 4501 Arlington Blvd Arlington VA 22203	OBG	O'REGAN, MD, Maureen 3802 N Fairfax Dr, #302 Arlington, VA 22203	OBG	SCHREINER, MD, David A. 2001 Columbia Pike Arlington VA 22204	IM
ENGH, MD, O Anderson 2465 Army-Navy Dr Arlington VA 22206	ORS	IOBAL, MD, M. Zalar 1715 N. George Mason Dr., #202 Arlington, VA 22205	OTO	MALPANI, MD, Kalidas D. 611 S Carlin Sprgs Rd, #401 Arlington VA 22204	IM	OKAIL, MD, Kamal K 4810 Beauregard St, #104 Alexandria, VA 22312	OTO	SCHWARTZ, MD, Raymond L 3231 Juniper Lane Falls Church VA 22044	PH
ERVINE JR, MD, Harry F. 6810 Fern Lane Annandale, VA 22003	OBG	JACKSON, MD, Charles B 251 Bliss Lane Great Falls, VA 22066	ORS	MANDANIS, MD, John P 1210 North Glebe Rd Arlington VA 22207	CD	OLIVER JR, MD, Cap H 2465 Army Navy Drive Arlington VA 22206	RHU	SHARMA, MD, Ashok, K 7438 Mason Lane Falls Church, VA 22042	R
ESPINEL, MD, Carlos H. 1715 N George Mason Dr, #401 Arlington, VA 22205	IM	JACOB, MD, Thomas N 3801 N Fairfax Dr, #402 Arlington, VA 22203	P	MARSHALL JR, MD, Jos K 2517 North Glebe Rd Arlington VA 22207	CD	ORLOSKY, MD, Albert J 4545 N 32nd Rd Arlington VA 22207	PD	SHEEHY, MD, Stephen Jos 5555 Columbia Pike Arlington VA 22204	GP
FENDER, MD, Gary R. 2465 Army-Navy Drive Arlington, VA 22206	IM	JASTRZEBSKI, MD, George W 4820 North 26th Street Arlington VA 22207	IM	MARTIN JR, MD, Lee Baldwin 4525 N 32nd St Arlington VA 22207	GS	ORSINGER, MD, Wm Hubert 5161 37th Street North Arlington VA 22207	GP	SHEIKH, MD, Mazhar U. 1228 Albebaran Drive Mc Lean, VA 22101	CD
FERRY, MD, Allen M 3713 S Geo Mason Dr, Apt 314 W Falls Church, VA 22041	ORS	JAYATE, MD, Rosy T 7821 Old Dominion Drive Mc Lean VA 22101	AN	MARTIN, MD, Lee B 3825 N Pershing Dr Arlington VA 22203	GP	PALMER, MD, Alfreed Monroe 200 South Oak Street Falls Church, VA 22046	GP	SHERIDAN, MD, Andrew J 1715 N George Mason Dr Arlington VA 22205	OPH
FINNERTY, MD, Paul Edward 1715 N George Mason Dr, #404 Arlington VA 22205	U	JUDSON, MD, John H 3801 N Fairfax Dr Arlington VA 22203	FP	MARTIN, MD, Shirley S 5205 Leesburg Pike Falls Church VA 22041	GYN	PALUMBO JR, MD, P M 8206 Leesburg Pike Vienna, VA 22180	ORS	SHUMAN, MD, Jos Elynn 1400 S Joyce St Arlington VA 22202	IM
FISHER, MD, Gerald John 5244 Dawes Ave Alexandria VA 22311	IM	KAKAVIATOS, MD, Nikos 3800 N Fairfax Drive Arlington VA 22203	IM	MC DONALD, MD, David B. 200 N Glebe Rd, #612 Arlington, VA 22203	IM	PANAGIS, MD, James S. 9812 Thunderhill Court Great Falls, VA 22066	ORS	SIBAY, MD, Hassan 3710 Oak Hill Way Fairfax, VA 22030	AN
FJORDBOTEN, MD, Ali Lee 3801 N Fairfax Dr Arlington VA 22203	OPH	KASENETZ, MD, Iver 6060 Arlington Blvd Falls Church, VA 22044	U	MC GAVIN, MD, Thos A 3801 N Fairfax Dr Arlington VA 22203	OBG	PARKER, MD, Kenneth R 601 S Carlin Sprgs Rd, #509 Alexandria, VA 22204	PM	SILEO, MD, Robert Peter 4141 N Henderson Rd Arlington, VA 22203	IM
FRANKLIN, MD, William George 1715 Geo Mason Dr, #107 Arlington, VA 22205	IM	KATZ, MD, Edward Lyle 3801 N Fairfax Dr Arlington VA 22203	NS	MC MAHON, MD, Geo Jos 8326 Epimard Ct Annandale VA 22003	P	PATTERSON, MD, Raymond F. 8 West Walnut Street Alexandria, VA 22301	P	SIMON, MD, Robert Isaac 5454 Wisconsin Ave, N.W. Chevy Chase, MD 20015	P
FRENKEL, MD, David Scott 3457 North 13th Street Arlington, VA 22201	P	KAUFMAN, MD, Paul 2786 N. Ouebec Street Arlington, VA 22207	PD	MC MANUS, MD, Reginald Paul 5300 Juxon Place Springfield VA 22151	IM	PEACOCK JR, MD, John H 3627 S 1st Rd Arlington VA 22204	PD	SIMS, MD, Arthur I 3801 N Fairfax Dr, #70 Arlington VA 22203	PD
FUCHS, MD, Glenn H. 7812 Butterfield Ln Annandale, VA 22003	D	KAYE, MD, Joseph T 2500 N Van Dorn St Alexandria VA 22302	ORS	MC MASTER, MD, Delphine A. 611 S Carlin Sprgs Rd, #102 Arlington, VA 22204	RHU	PERMAN, MD, Gerald P 2141 K St, Nw, #303 Washington, D.C 20037	P	SMITH, MD, Bernard Francis 1568 Forest Villa Lane Mc Lean VA 22101	R
GALLINER, MD, Wilfred Ernest 3801 N Fairfax Dr Arlington VA 22203	IM	KELLY JR, MD, Timothy L 1715 N Geo Mason Dr, #404 Arlington VA 22205	U	MC WILLIAMS, MD, Thos G 8306 Summerwood Drive Mc Lean VA 22102	R	PETERS, MD, Leslie Lawrence 1700 N Moore St, #925 Arlington VA 22209	US	SMITH, MD, Chas Glenn 4615 Lee Highway Arlington VA 22207	IM
GASPAR, MD, Maurice L. 820 N Stafford St Arlington, VA 22203	OPH	KIESEL, MD, Robert D 5015 Lee Hwy Rm 102 Arlington VA 22205	OPH	MCCABE, MD, Thomas Ambrose 1515 Chain Bridge Road McLean VA 22101	IM	PETTEY, MD, Thomas D. 3800 N. Fairfax Dr Arlington, VA 22203	OBG	SNYDER, MD, Bertram C 611 S Carlin Sprgs Rd, #512 Arlington VA 22204	IM
GAYDOS, MD, Lawrence Alfre 4141 N Henderson Road Arlington VA 22203	IM	KIM, MD, Hie Chul 8925 Burke Lake Road Springfield, VA 22151	PM	MCCRATH, MD, Francis J. 2517 North Glebe Rd Arlington, VA 22207	IM	PETTRONE, MD, Frank A 1715 N George Mason Dr, #504 Arlington VA 22205	ORS	SOLANO, MD, Simon 1715 N George Mason Dr, #305 Arlington VA 22205	OBG
GOALD, MD, Harold Jerome 4600 King Street Alexandria, VA 22302	NS	KING, MD, Peter Gabriel 3214 Lothian Rd, #103 Fairfax, VA 22031	PTH	WEISTER, MD, Robert Jay 8501 Aragon Lane Chevy Chase, MD 20015	HEM	PIERCE, MD, Phillip F. 4731 N. 23rd Street Arlington, VA 22207	ID	SOLET, MD, Leo 3708 N Woodstock Rd Arlington VA 22207	GP
GOLDMAN, MD, William D. 1715 N George Mason Dr, #205 Arlington, VA 22205	PD	KOHN, MD, Gary Marshall Nat'l Airport, Hqr #3 Washington, D.C 20001	AM	MERLINO, MD, Robin Beth 2465 Army Navy Drive Arlington, VA 22206	IM	PRICE, MD, Weldon A 6920 Holsing Lane Mc Lean VA 22101	PD	STALLINGS JR, MD, James H 107 North Virginia Ave Falls Church VA 22046	PD
GOLDSTEIN, MD, Samuel S. 2020 F St, Nw, Apt 926 Washington, DC 20006	IM	KOLIA, MD, Gulam-Mohmed M 6319 Castle Place Falls Church, VA 22044	IM	MIKSZEWSKI, MD, Jerold 611 S Carlin Springs Rd, #208 Arlington, VA 22204	N	PRINZ, MD, Werner 6240 Lakeview Dr Falls Church, VA 22041	GP	STAY, MD, Ellsworth J. 1701 N George Mason Dr Arlington, VA 22205	PTH
GONDOS III, MD, Zoltan 3260 Wilson Blvd Arlington VA 22203	GP	KORN, MD, Robert S. 3537 S. Stafford St Arlington, VA 22206	PD	MITCHELL, MD, Arhur V 4821 31st St South Arlington VA 22206	GP	PUZAK, MD, Michael August 4620 N 32nd Road Arlington VA 22207	U	STEINBERG, MD, Richard A. 3070 S. Abington St Arlington, VA 22206	EM
GONDOS, MD, Gordon Morris 9200 Alyssum Way Annandale, VA 22003	CHP	KOTH, MD, Douglas R 4000 N Ridgeview Road Arlington VA 22207	GS	MOHAPI, MD, Edith 7811 Antiopti Street Annandale, VA 22003	PD	RANGEL, MD, Jorge Garcia 4919 Wheatstone Dr Fairfax VA 22030	GP	STOKER, MD, Marlin Lawrence 5015 Lee Highway Arlington VA 22207	IM
GRANT, MD, Kathryn E. 6278 N. 15th Place Arlington, VA 22205	PM	LADY, MD, Wm Thurston 1505 Sombro Blvd Marathon, FIA 33050	GYN	MONDALL JR, MD, Philip 5201 Leesburg Pike, #102 Falls Church, VA 22041	PM	RAPP, MD, Michael T. 2002 Lorraine Ave Mc Lean, VA 22101	EM	STONE III, MD, Wm Leete 829 N Lincoln St Arlington VA 22201	IM
GREENBLUM, MD, Lucie 3801 Fairfax Dr, #61 Arlington, VA 22203	P	LANDIS, MD, Glen Austin 1701 North George Mason Drive Arlington VA 22205	NM	MOON, MD, Sung Kil 5411 Mt Greenview Court Burke, VA 22015	AN	REDLICH, MD, Michael 1077 30th Street, Nw Washington, D.C 20007	HEM	STRAWINSKY, MD, Eliz R Caro 3302 Galloway Road Falls Church VA 22042	P
GRUVER, MD, Robert H. 611 S. Carlin Sprgs Rd, #301 Arlington VA 22204	IM	LANG, MD, Edward Roberts 6305 Castle Place Falls Church VA 22044	NS	MORJARIA, MD, Mukund M 613 Castle Pl, #2-A Falls Church, VA 22044	IM	REINHARDT, MD, Erich Manfred 125 S Pitt Street Alexandria VA 22314	P	SUBHASH, MD, Shree 611 S. Carlin Springs Rd Arlington, VA 22204	GP
GURNEY, MD, Ronald Edward 1313 Dole Madison Blvd, #207 Mc Lean, VA 22101	IM	LATVEN, MD, K Chas 4375 A-Lee Highway Arlington VA 22207	OBG	MORRISSEY, MD, Wm Fitzgerald 2001 Columbia Pike Arlington VA 22204	IM	RICKERICH, MD, Chas L 611 S Carlin Sprgs Rd, #503 Arlington VA 22204	GS	SWAIN, MD, Garrett M 3039 N Peary Street Arlington VA 22207	NS
HACKMAN, MD, Helen M 9127 Glenbrook Road Fairfax, VA 22031	PH	LICAMELE, MD, William Louis 6208 Nethercombe Ct Mc Lean, VA 22101	CHP	MOSELY, MD, Linda Hays 2500 N Van Dorn St, #108 Alexandria, VA 22302	PS	RODILOSSO, MD, Philip Thos 1400 S Joyce St Arlington VA 22202	IM	TABOR, MD, Blanche 521 N. Lincoln St Arlington VA 22201	GP
HANNAN, MD, Chas Edmund 3801 N Fairfax Ave Arlington VA 22203	CRS	LICHTMANN, MD, Albert Laszlo 1104 Dunaway Drive Mc Lean VA 22101	AN	WOTT, MD, Howard O 829 N Lincoln St Arlington VA 22201	IM	ROMNESS, MD, Joseph O 1715 N George Mason Dr, #108 Arlington VA 22205	ORS	TERLINSKY, MD, Alan S. 611 S Carlin Sprgs Rd, #504 Arlington, VA 22204	IM

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THINT, MD, Ivy 611 S Carlin Sprgs Rd, #304 Arlington, VA 22204	IM	CAULKINS JR, MD, Chas W 220 Rosser Ave Waynesboro VA 22980	GS	KAPPES JR, MD, Wm Carl 13th At Magnolia Waynesboro VA 22980	PD	SPROUL, MD, George Thomas Post Office Box 2665 Staunton, VA 24401	PD	BAXTER, MD, Robt Francis Grundy Hospital Inc Grundy VA 24614	FP
TOCZEK, MD, Adriane E Mayakis 6319 Olmi-Landri Drive Alexandria, VA 22307	PD	COLEMAN, MD, Richard L M Staunton Medical Center Staunton VA 24401	OBG	KEIM, MD, Melvin N Staunton Med Center Staunton VA 24401	FP	STATHOS JR, MD, John A 2010 N Augusta Street Staunton, VA 24401	OPH	BERRY, MD, Bradley D Grundy Hosp Grundy VA 24614	GP
TOCZEK, MD, Stanislaw K 6319 Olmi-Landri Drive Alexandria, VA 22307	NS	CONCA, MD, Dominick Michael King's Daughters' Hospital Staunton, VA 24401	R	KLUGE, MD, Robt Carter Staunton Med Ctr Staunton VA 24401	PD	STIREWALT, MD, John Miles 112 Country Lodge Road Waynesboro VA 22980	PH	GREGORIOU, MD, Panos Geo Box 557 Clintwood VA 24228	GP
TOMLINSON, MD, H Evangeline 5249 Duke St, #11 Alexandria VA 22304	OBG	COOK, MD, Irving Kenneth Rt 2, Box 370 Waynesboro VA 22980	DR	LAWHORNE, MD, Larry Wayne 27 Taylor Street Staunton, VA 24401	GP	TALBERT, MD, John Robt 428 S Magnolia Ave Waynesboro VA 22980	OBG	LYLE, MD, Lurton Braxton Dickenson Clinic Box 557 Clintwood VA 24228	FP
TOUSTER, MD, Michael David 4141 N Henderson Rd Arlington VA 22203	IM	CRAWFORD, MD, David J. 1305 13th St Waynesboro VA 22980	FP	LEGETT, MD, John Albert Professional Center Waynesboro, VA 22980	PD	TENNEY JR, MD, Malcolm 24 Woodland Dr Staunton VA 24401	PH	MC DONALD, MD, Thos D Grundy Hospital Grundy VA 24614	PD
TURKEKUL, MD, Fuat 6700 Deland Dr Springfield VA 22152	AN	DARRACOTT, MD, Mixon Milford Staunton Med Ctr Staunton VA 24401	OBG	LOESCH, MD, Beverly Jean 220 Rosser Ave Waynesboro VA 22980	IM	THOMAS JR, MD, John H Greenville VA 24440	GP	MUCKENHAUSEN, MD, Christa P O Box 431 Pikeville, KY 41501	N
VADNEY, MD, Richard Claude 3801 N Fairfax Dr Arlington VA 22203	R	DEACON, MD, James Douglas 615 Oak Lane Waynesboro VA 22980	AN	LONG, MD, James Arthur 428 Maple Ave Waynesboro VA 22980	PUD	TIMS, MD, Roger Dean 809 Fairway Waynesboro, VA 22980	EM	PATEL, MD, Rajni P O Box 762 Grundy, VA 24614	PD
VANCE JR, MD, John Clair 5029 Millwood Lane NW Washington DC 20016	R	DEGEN, MD, Douglas B. Staunton Med Ctr Staunton, VA 24401	IM	MAC ILWAINE, MD, Wm Andrew 220 Rosser Ave Waynesboro VA 22980	IM	TODD III, MD, John W 40 Lambert St Med Ctr Staunton VA 24401	GS	PENN, MD, Thos Jefferson Box 992 Grundy VA 24614	GS
VERGARA, MD, Altonso 5248 Dawes Ave Alexandria VA 22311	IM	EDSON, MD, Ralph Howard 828 Meadow Brook Rd Waynesboro VA 22980	GS	MAHNESMITH, MD, Randolph C. 3 Hickory Hill Lane Fishersville, VA 22939	OBG	TOOMY, MD, Wm Nicholas The Professional Bldg Waynesboro VA 22980	IM	SANDERS, MD, Ulvert Ottway P O Box 94 Big Rock, VA 24603	GP
VOUVALIS, MD, Geo Sakellarios 6244 Williamsburg Blvd Arlington VA 22207	OPH	EGLESTON, MD, Du Bose 564 Oak Avenue Waynesboro VA 22980	OPH	MANNING, MD, Preston Cocke Staunton Medical Center Staunton VA 24401	GS	TYLER, MD, David 112 Pelham Drive Waynesboro VA 22980	OBG	SUTHERLAND, MD, Joshua P Rd 2 Box 64 B Grundy VA 24614	GP
WALKER, MD, Thomas M 1715 N George Mason Dr Arlington, VA 22205	ORS	ELLIOTT, MD, Constantine 1320 Ohio Street Waynesboro, VA 22980	P	MARTIN, MD, Gerry David 428 South Magnolia Ave Waynesboro, VA 22980	OPH	WEBSTER, MD, David K 44 Lambert St Ste 12 Staunton VA 24401	ORS	<b>CHARLOTTE</b>	
WALTHALL III, MD, David B. 1519 N McKinley Road Arlington, VA 22205	GP	FORBES III, MD, John Wm 113 First Street Staunton Draft VA 24477	GP	MC KIBBIN, MD, Douglas W. 220 Rosser Avenue Waynesboro, VA 22980	GS	WEEMS, MD, Bliss King P O Box 70 Waynesboro VA 22980	GS	AILS WORTH JR, MD, Robt Dea Ailsworth Med Ctr Keysville VA 23947	GP
WARE, MD, Robt Edward 611 S Carlin Sprgs Rd, #204 Arlington, VA 22204	U	FOSTER, MD, James Stephenson 24 Upper County Road South Dennis, MasS 02660	GP	MENK, MD, Karl F P O Box 2188 Staunton VA 24401	CLP	WILLIAMS, MD, Bernard Moore P O Box 152 Fishersville, VA 22939	P	DAVIS, MD, Charles Stewart P O Box 368 Drakes Branch VA 23937	GP
WEIMER, MD, Clarence J 2061 Woodstock St, Apt 202 Arlington, VA 22207	GP	GARDNER, MD, Richard Ernest 2010 N Augusta Street Staunton VA 24401	OPH	MILLER, MD, Richard B 113 First Street Staunton Draft, VA 24477	FP	WILLIAMS, MD, Hazael Jos 24 Terry Ct Staunton VA 24401	FP	DUNN, MD, J Wayland P O Box 366 Charlotte C H VA 23923	GP
WEYL, MD, W Leonard 611 S Carlin Sprgs Rd, #514 Arlington, VA 22204	GS	GATZEK, MD, Werner John E I Du Pont De Nemours Waynesboro VA 22980	OM	MORRIS, MD, Albert W 309 Sherwood Avenue Staunton VA 24401	AN	WINSTON, MD, Stephen Lee P O Box 3075 Staunton, VA 24401	AN	TUGGLE, MD, M. Stuart W. Route 2, Box 198 Keysville VA 23947	GP
WHITE, MD, Arthur E 9512 Mt Vernon Landing Alexandria, VA 22309	PM	GAYLORD, MD, Chas F 410 Mountainview Dr Staunton VA 24401	GP	NATHE, MD, James Edward 1070 Fairway Dr Waynesboro VA 22980	DR	WITT, MD, Nancy May Garrett P O Box 265 Fishersville VA 22939	P	<b>CHESAPEAKE</b>	
WHITE, MD, Robert M 1715 N George Mason Dr, #201 Arlington VA 22205	ORS	GILLESPIE, MD, Albert R 151 Woodland Drive Staunton VA 24401	OPH	NOLLEY, MD, Eugene Davis P O Box 100 Churchville VA 24421	FP	WOODS, MD, Paul A 362 S Laurel Ave Waynesboro VA 22980	FP	AULICINO, MD, Pat Louis 200 Medical Parkway, #210 Chesapeake, VA 23320	HS
WHITE, MD, Ruth M 4625 Old Dominion Dr Arlington VA 22207	GP	GILLESPIE, MD, James E. 560 Oak Avenue Waynesboro VA 22980	OPH	O HANLAN, MD, Jos Treacy 220 Rosser Ave Waynesboro VA 22980	GS	<b>BEDFORD</b>		BARAKKEY, MD, Amiele 110 Wimbeldon Sq, B Chesapeake, VA 23320	GS
WHITE, MD, Wm James 5275 Lee Hwy Arlington VA 22207	GP	GORSUCH, MD, Thos L The Professional Center Waynesboro VA 22980	IM	OGLE, MD, Rosa Christiane 625 Rosser Ave Waynesboro VA 22980	CLP	BRECHTELSBAUER, MD, David A Village Family Physicians Moneta VA 24121	FP	BROWN, MD, William P 2006 Old Greenbrier Rd #5 Chesapeake, VA 23320	AN
WIJETILLEKE, MD, Padma 6287 Dunaway Court Mc Lean, VA 22101	PD	GOTICO, MD, Rustico Tolentino 533 Galveston Rd Fredericksburg VA 22401	GP	ORR, MD, Wm King Box 449 Fishersville VA 22939	GP	BUCHANAN, MD, Brian D 1700 Whitfield Drive Bedford, VA 24523	IM	BRUNO JR, MD, Alphonse H L 202 De Paul Medical Bldg Norfolk VA 23505	OBG
WILLIAMS, MD, Austin Thos 6201 Leesburg Pike, #200 Falls Church VA 22044	IM	GRAHAM, MD, Sam D 2013 N Augusta St Staunton VA 24401	U	PAINTER, MD, Wm Graham Po Box 8 Fort Defiance VA 24437	GP	COHEN, MD, Leonard Joel 1700 Whitfield Dr Bedford VA 24523	FP	BUCHANAN, MD, Robt James 1324 Joliff Rd Chesapeake VA 23321	GP
WILLIAMS, MD, Thos Frasier 8350 Queensboro Drive Mc Lean VA 22102	OTO	GREY, MD, Wm Hugh 1102 N Coalter Street Staunton VA 24401	P	PAULY, MD, Charles Roman 220 Rosser Avenue Waynesboro VA 22980	D	DARDEN JR, MD, Oscar B 1700 Whitfield Dr Bedford VA 24523	IM	CANTOW, MD, Edward Francis 4037 Taylor Road Chesapeake VA 23321	ON
WISE JR, MD, Alan 1715 N Geo Mason Dr Arlington VA 22205	GS	GUSS, MD, John H 22 Orchard Road Staunton VA 24401	CD	PERKINS, MD, Paul Hening 428 S Magnolia Avenue Waynesboro VA 22980	OBG	HUTCHISON, MD, Wayne Thomas 1700 Whitfield Drive Bedford, VA 24523	FP	CASTANEDA, MD, Alberto J 1124 Green Run Square Virginia Beach, VA 23452	FP
WOLFSOHN, MD, Alice 5555 Columbia Pike Arlington VA 22204	GP	HALL III, MD, Snowden C 42 Lambert Street Staunton VA 24401	IM	POWERS, MD, Paxton P P O Box 2007 Staunton VA 24401	R	JENNINGS, MD, Eileen Thorpe 1700 Whitfield Dr, Box 889 Bedford VA 24523	FP	CASTILLEJO, MD, Raymundo A. 2006 Old Greenbrier Rd Chesapeake, VA 23320	AN
ZIMMET, MD, Steven Michael 1400 South Joyce St Arlington VA 22202	PUD	HAMMERBERG, MD, Jon Robert 501 Oak Avenue Waynesboro VA 22980	EM	RAU, MD, Ronald W 1151 13th St Waynesboro VA 22980	ORS	JENNINGS, MD, Thos H 1700 Whitfield Dr Bedford VA 24523	FP	COCKE, MD, John Alexander 736 Battlefield Blvd Chesapeake VA 23320	DR
ZINN, MD, Edward 1046 Baills Hill Rd Mc Lean VA 22101	P	HANNA JR, MD, H Michael Box 8 Ft Defiance VA 24437	IM	RICHARDSON, MD, Peter Bruce Staunton Medical Center Staunton, VA 24401	OPH	JENNETTE, MD, Freeman Wesley 1700 Whitfield Drive Bedford VA 24523	FP	CREEF, MD, J W 1201 Jackson St Chesapeake VA 23324	FP
ZOHN, MD, David Arthur 5201 Leesburg Pike, #102 Falls Church, VA 22041	PM	HANNA, MD, Stevan T P O Box 2157 Staunton, VA 24401	OTO	ROBERTSON JR, MD, Alex F 24 Ridgeview Rd Staunton VA 24401	IM	LUTH, MD, Janice Elaine Route 2, Box 25 Moneta, VA 24121	FP	CROSBY, MD, James Foster 318-D Battlefield Blvd, S Chesapeake VA 23320	FP
<b>AUGUSTA</b>		HARMAN, MD, W.E 901 Blue Ridge Dr Staunton VA 24401	PD	ROGERS, MD, William E. 362 S. Laurel Ave Waynesboro VA 22980	DR	RHODES JR, MD, Hebert Paul Bedford Memorial Hosp Bedford VA 24523	DR	DAJAO, MD, Rogaciano M. 1105 Halifax Ave Portsmouth VA 23703	GP
ANDERSEN, MD, Charles F 1151 13th St Waynesboro VA 22980	ORS	HARPER, MD, Forest G P O Box 2007 Staunton VA 24401	R	SACHNO JR, MD, Roman Staunton Medical Center Staunton VA 24401	IM	ROBINSON JR, MD, Dennis H 309 N Bridge St Bedford VA 24523	IM	DONLAN JR, MD, Charles J. 250 W Brambleton Ave, #201 Norfolk, VA 23510	IM
ANDERSON, MD, J Powell P O Box 70 Waynesboro VA 22980	FP	HARRISON III, MD, Carrington 1524 N. Augusta Street Staunton VA 24401	ORS	SACKS, MD, Eugene Ira 1298 Sunset Lane Waynesboro VA 22980	GS	RUCKER JR, MD, Saml L Moneta VA 24121	GP	DOWNS, MD, Edward Jay 3352 Prince Of Wales Court Virginia Beach VA 23454	EM
BAIN, MD, James Britton 465 Cherry Ave Waynesboro VA 22980	OPH	HEATWOLE, MD, John Paul P O Box 992 Waynesboro VA 22980	AN	SHERRY, MD, John Barry Waynesboro Community Hosp Waynesboro VA 22980	PTH	RUCKER, MD, Wm Vincent Bedford VA 24523	GP	DU PUY, MD, Theodore E. 3233 Bruin Drive Chesapeake, VA 23321	ORS
BELL, MD, Rudolph Mardre Staunton Med Ctr Lambert St Staunton VA 24401	ABS	HEATWOLE, MD, Stanley E. 304 College Circle Staunton VA 24401	EM	SHIELDS JR, MD, Randolph T 36 Ridgeview Road Staunton VA 24401	GS	WENGER JR, MD, John Robert 1700 Whitfield Dr Bedford VA 24523	IM	GIANNOTTO, MD, Richard P. 501 Battlefield Blvd, N Chesapeake, VA 23320	ORS
BELL, MD, Thos Grasty Staunton Med Ctr Staunton VA 24401	OBG	HIGGS, MD, James Albert Med Center Ste 11 Staunton VA 24401	GP	SMITH, MD, Mc Kelden Staunton Med Ctr Staunton VA 24401	IM	WENTE JR, MD, John Anthony 811 Oak St Bedford VA 24523	GS	GOLDMAN, MD, Edwin E. 3800 Weiss Lane Chesapeake, VA 23323	EM
BOATRIGHT, MD, Kenneth A. 1151 13th Street Waynesboro, VA 22980	ORS	HOFFMAN, MD, Michael A. P O Box 2856 Staunton, VA 24401	P	SMITH, MD, Wm Crenshaw 220 Rosser Ave Waynesboro VA 22980	PD	WOODROOF, MD, Kerry C. 412 North Bridge St Bedford, VA 24523	OPH	GONZALES, MD, Jose E. 509 Depaul Med Bldg Norfolk, VA 23505	U
BRANSCOME, MD, William C Staunton Medical Center Staunton VA 24401	IM	HOLMES, MD, I Earl 222 Barracks Beach Dr Colonial Beach, VA 22443	R	SOWERS, MD, William F Medical Center Staunton, VA 24401	D	<b>BOTETOURT</b>		HABEEB, MD, Edward David 501 Battlefield Blvd, N Chesapeake, VA 23320	ORS
BRYAN JR, MD, Phillips R. 2013 North Augusta St Staunton, VA 24401	U	HOWLETT, MD, Stephen Andrew 13 Th At Magnolia Waynesboro VA 22980	GE	SPROUL, MD, A Erskine 41 Woodland Dr Staunton VA 24401	PTH	<b>BUCHANAN-DICKENSON</b>		JAMALI, MD, Alireza 110 Wimbeldon Sq, #C Chesapeake, VA 23320	ORS
						ALDERMAN, MD, Kurtz Edward Dickenson Clinic, Box 557 Clintwood VA 24228	GP	JENNINGS, MD, W Stanley 1160 Virginia Ave Chesapeake VA 23324	GP

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KAGAN, MD, Larry Howard 1012 Macdonald Road Chesapeake, VA 23325	FP		<b>CULPEPER</b>	ANDREWS JR., MD, Michael J. P O Box 1360 Danville, VA 24543	U		HARVEY, MD, Herbert Whitley 344 Ross St Danville, VA 24541	GP	SHIFLETT, MD, Douglas W. 101 Holbrook Street Danville, VA 24541	GE	
KANTER, MD, Hubert J 200 Medical Parkway, #205 Chesapeake VA 23320	GS		ALLEN, MD, Benjamin F 1051 Oaklawn Drive Culpeper, VA 22701	ORS			HARVIE, MD, Edwin James 101 Holbrook Street Danville VA 24541	IM	SILVERMAN, MD, Herbert R 130 Gray Street Danville VA 24541	GS	
KUEHN, MD, Hubert W Chesapeake Gen Hosp Chesapeake, VA 23320	R		BATES JR., MD, Harry Clark P O Box 368 Rixeyville, VA 22737	OM			HOFFMAN JR., MD, Allan A P O Box 1360 Danville VA 24543	U	SIMMONS, MD, Robt Geo 101 Holbrook St Danville VA 24541	OBG	
LAIBSTAIN, MD, Robert B 1012 Macdonald Road Chesapeake, VA 23325	FP		BROWMAN, MD, Geo Ellis P O Box 550 Culpeper VA 22701	GS			HOSKINS, MD, Horace D 231 Parkview Dr Danville VA 24541	OPH	SOYANGCO, MD, Alfredo Lopez 142 Major Court Danville VA 24540	AN	
MASTER, MD, James Monroe 506 S Independence Blvd, #202 Virginia Beach, VA 23452	P		CATE, MD, L Huntley Brightwood VA 22715	GP			JOSEF-GUANZON, MD, Pat 130 Gray Street Danville VA 24541	PD	SPAINHOUR JR., MD, Jack Bryan 101 Holbrook Danville VA 24541	GE	
LEAVY JR., MD, Philip G. 1125 Westover Ave Norfolk, VA 23507	EM		CAVE, MD, Wm Belfield Box 347 Madison VA 22727	FP			KOPLER, MD, Julian Arthur 115 South Main Street Danville, VA 24541	IM	SPRINKLE, MD, James Dean 171 S Main St Danville VA 24541	GS	
LEGASPI, MD, Amante G 4424 Moosewood Drive Virginia Beach, VA 23462	AN		CHEFETZ, MD, Richard Alan P O Box 386 Madison, VA 22727	FP			KUHN, MD, Robt Anthony P O Box 603 Clifton Forge, VA 24422	N	STARLING JR., MD, James F P O Box 1360 Danville VA 24543	NEP	
LEGUM, MD, Larry L. 113 Coastal Way Chesapeake, VA 23320	D		CHILES III, MD, Morton Perrin 1222 Blue Ridge Ave Culpeper VA 22701	FP			LAIRD, MD, Thos Kerr 990 Main St Danville VA 24541	GS	STONEBURNER, MD, John M 171 S Main St Danville VA 24541	GS	
LOXLEY, MD, Sidney S 501 Battifield Blvd, N Chesapeake, VA 23320	ORS		COOK, MD, J. Bryon 1043 Oaklawn Drive Culpeper, VA 22701	IM			LANDES, MD, Ralph Roy 1040 Main St Danville VA 24541	U	THOMPSON JR., MD, Girard V Box 570 Chatham VA 24531	GP	
MAGIER, MD, Igor 200 Medical Pkwy, #313 Chesapeake, VA 23320	P		CRAMER, MD, Alfred Bartlett P O Box 69 Culpeper VA 22701	GE			LISSITER, MD, Max Errington 181 S Main St Danville VA 24541	PD	THOMPSON, MD, Girard Vaden Box 570 Chatham VA 24531	FP	
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FUSCO, MD, Frank Danl 8318 Arlington Blvd, #303 Fairfax, VA 22031	PUD	GRUNDEHNER, MD, Marietta 4401 Wakefield Drive Annandale, VA 22003	NEP	HERSHBERG, MD, Sandra Gail 5225 Conn Ave, Nw, #705 Washington, DC 20015	CHP	KAY, MD, George Gordon 10721 Main Street Fairfax VA 22030	IM	LANTER, MD, David Lloyd 1515 Chain Bridge Rd Mc Lean VA 22101	OPH
GAERTNER JR., MD, Richard L 307 Maple Ave W Vienna VA 22180	ORS	GUDNASON, MD, Halldor Viktor Box 1417, Suite A-41 Alexandria, VA 22313	AN	HEYL, MD, Petar S. 3300 Gallows Road Falls Church, VA 22046	OBG	KEIM, MD, Daniel E. 5220 Rolling Road Burke, VA 22015	PD	LARSON, MD, Steven D. 2579 John Milton Dr, #200 Herndon, VA 22071	FP
GALKIN, MD, Lloyd 2830 Flagmaker Drive Falls Church, VA 22042	GP	GUILLAUME, MD, Robt L 313 Park Ave Falls Church VA 22046	IM	HILL, MD, Elizabeth Harman 10721 Main St Fairfax VA 22030	IM	KELLY, MD, John Francis 5262 Dawes Ave Alexandria VA 22311	PD	LASTER, MD, James 6060 Arlington Blvd Falls Church VA 22044	GP
GALLAGHER, MD, Edward James 10090 Main St Fairfax VA 22030	IM	GUILLEN, MD, Manuel 8303 Arlington Blvd, #201 Fairfax, VA 22031	A	HILLIARD, MD, Janet Karen 3300 Gallows Rd Falls Church VA 22046	PD	KENDERS, MD, Kathryn L 5803 Manchester Pl Nw Washington DC 20001	OS	LATKIN, MD, Patar Chas 6201 Leesburg Pike, #300 Falls Church VA 22044	PDA
GANNON, MD, James A. 729 Hillcrest Dr Staunton, VA 44401	GP	GURNEY, MD, Robert Waring 11315 Sunset Hills Road Reston, VA 22090	D	HIRSCHMAN, MD, Bernardo 8303 Arlington Blvd, #205 Fairfax VA 22031	P	KENNEDY, MD, Carol Elizabeth 8316 Arlington Blvd Fairfax, VA 22031	IM	LAWRENCE, MD, Mark Allen 8612 Tebbes Lane McLean, VA 22102	P
GARCIA, MD, Alberto J 8516 Leesburg Pk #3 Vienna VA 22180	OBG	GUTIERREZ, MD, Jose Antonio 1515 Chain Bridge Rd Mc Lean VA 22101	GE	HODIN, MD, Earl 3301 Woodburn Road, #205 Annandale, VA 22003	PDS	KENNEDY, MD, Stephen F 1429 Highwood Drive McLean, VA 22101	AN	LAZARTE, MD, Raul A 3300 Gallows Rd Falls Church VA 22046	PD
GARCIA, MD, Ramon 3156 Holmes Run Rd Falls Church VA 22042	AN	GUYNN, MD, Cyrus Harding 8344 Trator Lane Springfield VA 22152	IM	HOGAN, MD, Martha L Wyrick 8316 Trator Lane Springfield, VA 22152	PD	KERMAN, MD, Shelly Lynn 11091 Main St Fairfax VA 22030	PTH	LE NARD, MD, Patar Dennis 1485 Chain Bridge Road Suite 202 Mc Lean, VA 22101	GS
GARCIA, MD, Raul R 153 Glyndon St S E Vienna VA 22180	FP	HAIR, MD, Joyce P 8316 Arlington Blvd, #520 Fairfax VA 22031	OBG	HOLT, MD, Ronald R 3300 Gallows Road Falls Church, VA 22046	AN	KESSLER, MD, Carl Paul 3545 Chain Bridge Road Fairfax VA 22030	ORS	LEARY, MD, Patrick J 410 Maple Ave W, #4 Vienna VA 22180	OBG
GARCIA, MD, Robt Courtney 12700 Watertown Court Potomac, MD 20854	R	HALFOND, MD, Ivan P O Box 1116 Centerville, VA 22020	IM	HORAN, MD, Michael Thomas 6120 Brandon Ave, #305 Springfield, VA 22150	P	KESSLER, MD, Chester Wm 4231 Markham St, #222 Annandale VA 22003	GP	LEBOWITZ, MD, J Martin 6845 Elm Street Suite 615 Mc Lean, VA 22101	U
GARDNER, MD, Allen Stiles 6807 Springfield Plaza Springfield VA 22150	IM	HALL, MD, Allan 6505 Dearborn Drive Falls Church VA 22044	GS	HORN, MD, Henry J 4540-A John Marr Drive Annandale VA 22003	FP	KHAN, MD, Mohammad Aqq 5216 Rolling Road Burke, VA 22015	IM	LEBUFFE, MD, Francis P 404 E Broad St Falls Church VA 22046	P
GASHI, MD, Faton 10513 Samaga Dr Oakton, VA 22124	PM	HALLAL, MD, Fadell Jos 8316 Arlington Blvd #524 Fairfax VA 22030	IM	HORN, MD, Martin Seth 10721 Main St, #304 Fairfax, VA 22030	D	KIERNAN, MD, Kavin W 3300 Gallows Road Falls Church, VA 22046	P	LEDERMAN, MD, Martin Edward 1515 Chainbridge Rd Mc Lean VA 22101	OPH
GAVERLOVICH, MD, Lillian 200 Little Falls St Falls Church VA 22046	IM	HALTERMAN, MD, Roger H 13204 Beall Creek Ct Potomac MD 20854	ND	HORNE, MD, Allen Bernard 6715 Whittier Avenue Mc Lean VA 22101	FP	KIESSLING, MD, Alice H 7048 Haycock Rd Falls Church VA 22043	PYA	LEE, MD, Chun Sheng 6013 Claiborne Dr Mc Lean VA 22101	OBG
GAZALE, MD, Wm J 10720 Main St Fairfax VA 22030	ORS	HAM JR., MD, Tibor John 135 Center Street, South Vienna, VA 22180	IM	HORTON, MD, Jack Donald 8316 Arlington Blvd Fairfax, VA 22031	CDS	KILFEATHER, MD, John E 8301 Arlington Blvd Fairfax, VA 22031	IM	LEE, MD, David Donghan 9823 Laurel Street Fairfax VA 22030	GP
GEHARA, MD, David J 6201 Leesburg Trnpke, #308 Falls Church VA 22044	P	HAM, MD, T. 135 Center St South Vienna VA 22180	GP	HOWE JR., MD, Allan K. 2579 John Milton Dr, #200 Herndon, VA 22071	FP	KIM, MD, Chungkook 9104 Trusler Court Manassas, VA 22110	OBG	LEE, MD, Kyung Ja Shin 3450 Beuregard St, #2 Alexandria, VA 22302	P
GELMAN, MD, Howard K 8316 Arlington Blvd Fairfax VA 22031	OTO	HANFUNG JR., MD, Carl 7501 Little River Turnpike Annandale VA 22003	FP	HUANG, MD, Amy Hwei-Mei 1445 Laurel Hill Rd Vienna VA 22180	PTH	KIM, MD, Mi Yong 3512 Barkley Drive Fairfax, VA 22031	OBG	LEE, MD, Sang Nam 3300 Gallows Road Falls Church, VA 22046	ON
GELTNER, MD, Jane Weinberg 2501 Parker Ln Alexandria VA 22306	CLP	HANNON, MD, John F. 2946 Sleepy Hollow Rd Falls Church VA 22044	OPH	HUBACH, MD, Frederick Willis 6715 Whittier Avenue Mc Lean VA 22101	FP	KIRKSHNER, MD, Louis Paul 102 Maple Ave, E, #1-A Vienna VA 22180	PD	LEE, MD, Won Ro 3450 N Beuregard St, #2 Alexandria VA 00000	CD
GEOLY, MD, Kenneth Lucian 8316 Arlington Blvd Fairfax, VA 22031	NEP	HARRER, MD, David S 6809 Glenclave Dr Clifton VA 22024	CLP	HUNT, MD, Robt Clarence 313 Park Ave Falls Church VA 22046	PD	KLAM, MD, Warren Peter P O Box 7201 Arlington, VA 22207	PD	LEFRACK, MD, Edward Arthur 3301 Woodburn Rd, #301 Annandale, VA 22003	TS
GHAEMI III, MD, Kamal Box 427 Mc Lean VA 22101	N	HARSANYI, MD, Paul Gabor 102 Maple Ave E, #1-A Vienna VA 22180	R	HUNTINGTON, MD, Daniele F 3300 Gallows Rd, Falls Church VA 22046	PD	KLAPPROTH, MD, Hans Joachim 7501 Little River Annandale VA 22003	U	LEFTON, MD, Charles Stuart 307 Maple Ave W, #J Vienna VA 22180	ORS
GILLANDERS, MD, Robt James 360 Maple Avenue West Vienna VA 22180	OBG	HARSHAW JR., MD, William Geo 3300 Gallows Rd Falls Church VA 22046	R	HURWITZ, MD, Byron Stuart 7314 Burdette Ct Bethesda MD 20817	OPH	KNERR, MD, Robt James 410 Maple W Vienna VA 22180	PD	LEGASPI, MD, Alfredo Lacuna 3300 Gallows Rd-Fairfax Hosp Falls Church VA 22046	AN
GISOLFI, MD, Roger Vincent 5001 Lone Oak Place Fairfax, VA 22032	PM	HART JR., MD, Richard Jos 6400 Arlington Blvd Falls Church VA 22042	IM	INGLEFIELD, MD, Jos Thacher 6329 Linway Terrace McLean, VA 22101	PDA	KNOX, MD, Henry Donald Springfield Prof Park 8316 Trator Lane Springfield VA 22152	PD	LEIDELMEYER, MD, Rainald 3405 St. Paul Place Fairfax, VA 22031	EM
GLOVER JR., MD, Wm Lloyd 10721 Main St Fairfax VA 22030	U	HARVAN, MD, David 9625 Surveyor Ct, #300 Manassas VA 22110	OPH	ISAAC, MD, Bernard Anthony 135 Leesburg Pike, #14 Sterling, VA 22170	OTO	KNUDSON, MD, Homer Ellsworth 8320 Old Courthouse Rd Vienna VA 22180	GYN	LEON, MD, Antonio Enrique 7005 Barkwater Court Bethesda MD 20817	AN
GODWIN, MD, Ira David P O Box 188 Fairfax VA 22030	PTH	HASKO, MD, Barbara Ann 4053 Olley Lane Fairfax, VA 22032	IM	IVY, MD, Michael Warren A 2761 Annandale Road, #2 Falls Church, VA 22042	OBG	KOEHLER, MD, Rolt Altrid 10721 Main St Fairfax VA 22030	IM	LESOWITZ, MD, Sidnay Allan 6319 Castle Pl Falls Church VA 22044	D
GOLDBERG, MD, Michael Harvey 8134 Old Keene Mill Rd, #300 Springfield, VA 22152	OPH	HATTWICK, MD, Michael A 6436 West Langley Lane Mc Lean VA 22101	GPM	JACKLIN, MD, Lawrence A 511 W Broad St Falls Church VA 22046	US	KOLVEREID, MD, Edward Ronald 8316 Arlington Blvd, #526 Fairfax, VA 22031	OBG	LESSIN, MD, Bruce Edward 1313 Dolly Madison Blvd, #207 Mc Lean VA 22101	IM
GOLDENBERG, MD, Robin Ira 8318 Arlington Blvd Fairfax VA 22031	PD	HAWKEN, MD, Samuel M 3301 Woodburn Road Annandale, VA 22003	ORS	JACOBSON, MD, Cecil Bryant 8320 Old Court House Road Vienna VA 22180	OS	KOONS, MD, Gregory Mark 11198 Lee Highway Suite B Fairfax VA 22030	OBG	LEVINE, MD, Leonard S 6807 Springfield Pl, #206 Springfield VA 22150	OBG
GOLDSTEIN, MD, Leonard Steven 3018 Williams Drive Fairfax VA 22031	P	HEAD, MD, Gordon Lawrence 11302 Lapham Drive Oakton, VA 22124	DR	JASON, MD, Casey John 6580 Braddock Road Alexandria, VA 22312	PD	KORNBLUTH, MD, Ralph Ross 8303 Arlington Blvd Ste 207 Fairfax VA 22031	PD	LEWIS, MD, Kerry Randall 11320 Lafferty Lane Fairfax, VA 22030	GP
GOLOMB, MD, Herbert S. 1910 Woodgate Lane Mc Lean VA 22101	D	HEATH, MD, John Francis 313 Park Ave Falls Church VA 22046	P	JOHN, MD, Sarah A 2857 Pine Spring Road Falls Church VA 22042	PD	KORNHAUSER, MD, Michael James 5597 Seminary Rd, #1706, S Falls Church VA 22041	P	LIBBY, MD, Russell C. 316 Arlington Blvd, #502 Fairfax, VA 22031	PD
GONZALES, MD, Federico Carlos 10525 West Drive Fairfax, VA 22030	IM	HEFTER, MD, Lawrence G 3300 Gallows Rd Falls Church VA 22046	PTH	JOHNSON JR., MD, Lester Dean 8301 Arlington Blvd, #305 Fairfax VA 22031	OBG			LICATA, MD, Robert M. 6800 Fleetwood Dr, #111 Mc Lean VA 22101	P



## 10 FAIRFAX

LINDE, MD, Richard Emil 6231 Leesburg Pike Falls Church VA 22044	OT *	MATTHEWS, MD, Robt Geo 3301 Woodburn Rd, #304 Annandale, VA 22003	CD	NEFF, MD, Allan J. 3300 Gallows Rd Falls Church VA 22046	AN	PINNAR, MD, Robt Lloyd 8317 Rlington Blvd, #226 Fairfax VA 22031	GS *	RIFAAT, MD, Monira K 8118 Good Luck Rd Lanham MD 20706	CLP
LINDSTEDT, MD, Jan Gustaf 1148 Marion Avenue McLean, VA 22101	DR *	MAY, MD, Russell Leon 8316 Arlington Blvd Ste 532 Fairfax VA 22031	OBG	NEIDLINGER, MD, Robt Waller 6120 Brandon Ave Springfield VA 22150	OPH *	PISTENMAA, MD, David A. 5812 Brookside Drive Chevy Chase, MD 20815	R *	ROATH, MD, Michael Steven 5411 Gov Yeardeley Dr Fairfax VA 22032	P *
LINEBERGER, MD, Adrian Smith 11091 Main Street Fairfax VA 22030	PTH *	MC ATEER, MD, Gerald H 3220 Cathedral Ave, NW Washington, DC 20008	GS *	NEVIASER, MD, Thomas J 360 Maple Ave. W Vienna, VA 22180	ORS *	PO, MD, Heng-Tsui 8700 Pembroke Ct Fairfax VA 22031	AN *	ROBECK, MD, Ilana Rae 3542 N Dinwiddie St Arlington, VA 22207	IM *
LITOVITZ, MD, Gary L. 8303 Arlington Blvd, #205 Fairfax, VA 22031	P	MC AVEYNE, MD, William J 8724 Evangel Drive Springfield, VA 22153	PD *	NGAU, MD, Curtis A. 8330 Old Courthouse Rd, #730 Vienna VA 22180	PS *	PODOLNICK, MD, Nelson 255 W Broad St Falls Church VA 22046	FP *	ROCHMS, MD, Ann Romatowski 8316 Arlington Blvd Fairfax VA 22031	P *
LITTLETON, MD, Philip Ray 6120 Brandon Ave Springfield VA 22150	PD	MC CANN, MD, Wm John 1307 Altamira Ct McLean VA 22101	OBG	NGUYEN, MD, Ouan O 4932 Americana Drive Annandale, VA 22003	IM	POLICELLI, MD, Vincent A 10028 Garrett St Vienna VA 22180	OBG *	ROCHMS, MD, Paul Gregor 10220 Katie Bird Lane Vienna, VA 22180	RHU *
LIVINGOOD, MD, J.K. 1051 Edden Street Herndon VA 22070	GP	MC CLURE, MD, Wm West 6060 Arlington Blvd Falls Church VA 22044	IM	NICKLAY, MD, James Thomas 6231 Leesburg Pike Falls Church, VA 22044	OTO *	POLLOCK, MD, Donald David 600 W Taylor Run Pky Alexandria, VA 22314	P	ROGERS, MD, Charles L. 611 S Carlin Sprgs Rd, #505 Arlington, VA 22204	ORS
LLANERAS, MD, Rene F 1712 Clubhouse Rd Reston VA 22090	PD	MC DOWALL, MD, James Douglas 3545 Chain Bridge Rd Fairfax VA 22030	PD	NIMS, MD, Linda Park 301 West Maple Ste 4 Vienna VA 22180	D *	PORETZ, MD, Donald Martin 8318 Arlington Blvd Fairfax VA 22030	ID *	ROLL JR, MD, William E 10721 Main St Fairfax VA 22030	GS *
LORIO, MD, Jos Philibert 4400 Newdale Rd Annandale VA 22003	P	MC FARLAND, MD, John W. 3842 North Dittmar Rd Arlington, VA 22207	P	NOEL, MD, Roger A. 8318 Arlington Blvd Ste 302 Fairfax VA 22031	IM	POTTER, MD, Michael Charles 8318 Arlington Blvd, #303 Fairfax, VA 22031	GS	ROMERO, MD, Gonzalo 611 S Carlin Sprgs Rd, #220 Fairfax, VA 22031	GS *
LOTZ, MD, Myron 301 Maple Ave W 3-A Vienna VA 22180	IM *	MC MURRER JR., MD, James P 316 Windover Ave Vienna VA 22180	CHP *	NOTES, MD, David Raymond 10721 Main St Fairfax VA 22030	OPH *	PRESSMAN, MD, Howard Ira 2210 Predella Drive Silver Spring, MD 20902	P	ROONEY, DO, Danl Dare 301 Maple Ave West Vienna VA 22180	OST
LUX, MD, Ann Mary 9327 Campbell Rd Vienna VA 22180	P	MCCABE, MD, Dennis J. 301 Maple Ave, W, #4-C-D Vienna VA 22180	R	NOVELLO, MD, Antonia C. 1315 31st Nw Washington, DC 20007	PD *	PRICE, MD, Neel J 6316 Castle Pl Falls Church VA 22044	OBG *	ROSEN, MD, Leonard A 8990 Fern Park Drive Burke, VA 22155	OBG
LYLES JR, MD, John Wm 7501 Littleliver Turnpike Annandale VA 22003	OBG *	MELLA, MD, Barbara A 3251 Old Lee Hwy Fairfax VA 22030	N *	NOWELL, MD, John Francis 611 S. Carlin Sprgs Rd, #404 Arlington, VA 22204	OPH *	PRIETO JR., MD, Danl Corneja 6200 Vernon Palmer Ct McLean VA 22101	R *	ROSENTHAL, MD, Richard R 4303 Ann Fitzhugh Dr Annandale VA 22003	A *
LYNCH, MD, George Michael 2579 John Milton Dr, #200 Herndon VA 22071	FP	MELMED, MD, Allan Stanley 7659 Leesburg Pike Falls Church VA 22043	P	O DONNELL, MD, Robt Jos 6319 Castle Pl Falls Church VA 22044	OS *	PROMINSKI, MD, John E 200 Little Falls St Falls Church VA 22046	R *	ROSENTHAL, MD, Steve 2510 Freetown Drive Reston VA 22091	P
MACKINTOSH, MD, Alan 3301 Woodburn Rd, #102 Annandale VA 22003	FP	MENSCH, MD, Arthur H 4320 Seminary Road Alexandria, VA 22314	PTH *	O NEILL, MD, Thos Michael 11705 Bowman Green Dr Reston VA 22090	D	PUGSLEY, MD, Louis Ouan 8316 Arlington Blvd, #506 Fairfax VA 22031	OBG *	ROSS, MD, Michael A. 6845 Elm St, #509 McLean VA 22101	OBG *
MACMANUS, MD, Quentin 3301 Woodburn Rd, #301 Annandale, VA 22003	CDS *	MERO, MD, James Hill 301 Maple Ave Ste 4C Vienna VA 22180	R	O'BRIEN, MD, Thos Edward 313 Park Ave Falls Church VA 22046	IM	PUJOL, MD, Jackie 431 Maple Ave., W. Vienna VA 22180	PD	ROSS, MD, Peter S 8301 Arlington Blvd, #205 Fairfax, VA 22031	EN
MAGANIAS, MD, Nicholas H 7520 Royal Oak Dr McLean VA 22102	PD *	METZGER, MD, Arthur Zelig 1538 Windy Hill Rd McLean VA 22102	PD	OVERHOFF, MD, Peter 1515 Chain Bridge Rd McLean VA 22101	OPH	PULIZZI JR., MD, John S 3450 North Beauregard Alexandria, VA 22302	US *	ROTH, MD, Richard Lee 6845 Elm St, #210 McLean VA 22101	P
MAGNANT, MD, Geo Arthur 5198 Dawes Ave Alexandria VA 22311	PD *	MEZA, MD, Rene S. 6319 Castle Place #C Falls Church, VA 22044	GP	OCAMPO, MD, Alexander T. 11091 Main Street Fairfax, VA 22030	P *	QUINNELL, MD, Robt Kay 380 Maple Ave W Vienna VA 22180	FP	ROUADY, MD, William A 5504 Backlick Road Springfield, VA 22151	NS *
MAILLIS, MD, Maxwell Sherwood 6807 Springfield Pl, #201 Springfield VA 22150	CD	MIKHAIL, MD, Eva Labib 222 Apple Blossom Court Vienna, VA 22180	PTH *	ODIAGA, MD, Carlos E 3301 Woodburn Road Annandale VA 22003	GP *	QUINTOS, MD, Eugenia J. 6328 Mapapple Place Alexandria, VA 22312	PTH	ROY, MD, Gaston E 7501 Little River Tpke, #306 Annandale VA 22003	P *
MAJOR, MD, Mary Jane 1715 N George Mason Dr, #204 Arlington, VA 22205	IM *	MILLER, MD, A Larry 311 Maple Ave, W, #H Vienna VA 22180	A *	OLDMIXON, MD, Willard J 2455 Army-Navy Dr Arlington VA 22206	PTH *	RAFAY, MD, Ernest Geo 6304 Waterway Dr Falls Church VA 22044	FP *	RUBIN, MD, Max Bernard 1515 Chain Bridge Rd McLean VA 22101	D
MALKA, MD, Jeffrey S 6845 Elm St McLean VA 22101	ORS	MILLER, MD, John Alfred 3301 Woodburn Rd, #206 Annandale, VA 22003	HEM	ONDER, MD, Mehmet Hami 3505 Cobb Dr Fairfax VA 22030	PUD *	RAFI, MD, Esmail 8301 Arlington Blvd Fairfax VA 22031	IM *	RUIZ, MD, Gil Madrigal 200 Little Falls Rd Falls Church VA 22046	IM
MAMANA, MD, John Philip 8560 Georgetown Pike McLean VA 22101	IM	MILLER, MD, Karen J. 6060 Arlington Blvd Falls Church, VA 22044	PD	ORMANDY, MD, Laszlo 6374 Lakeview Drive Falls Church, VA 22041	ORS *	RALPH, MD, Robt D 716 East Broad St Falls Church VA 22046	OTO *	RUSSO, MD, Eugene P. 737 Walker Rd, #4-A Great Falls VA 22066	GS *
MANALO, MD, Bayani L. 6400 Seven Corners Pl Falls Church, VA 22044	IM	MOINFAR, MD, Mohamad Reza 10721 Main Street, #2100 Fairfax VA 22030	PD	OSHINSKY, MD, Arnold L. 6060 Arlington Blvd Falls Church VA 22044	OPH *	RAMASWAMY, MD, Isabel Jamili 820 North Stafford St Arlington, VA 22203	PD	RUSSO, MD, Voislava C. 8012 Old Falls Road McLean, VA 22107	PD *
MANDES, MD, Thomas C. 370 West Maple Ave, #3 Vienna, VA 22180	IM	MONIZ JR., MD, Alberi 6060 Arlington Blvd Falls Church VA 22044	PD	OSSOFKY, MD, Helen Johns 1333 Merrie Ridge Rd McLean VA 22101	CHP *	RATNER, MD, Richard A. 5210 Elliott Rd Bethesda, MD 20816	P	RYAN, MD, Mary C 1423 Aldenham La Reston VA 22090	PD *
MANGOLD, MD, Harry Armstrong 8318 Arlington Blvd, #305 Fairfax, VA 22031	A *	MOORE, MD, Ralph Wm 5101-C Backlick Road Annandale VA 22003	P *	OVERTON, MD, Eugene W. 11200-A Lee Highway Fairfax, VA 22030	FP *	REA, MD, Edward L 200 Little Falls Street Falls Church VA 22046	IM	SABELLA, MD, Donald A 70 West Cornwall Street Leesburg, VA 22075	IM
MANGUIKIAN JR., MD, Dertad 9936 Main St Fairfax VA 22031	OPH	MORALES, MD, Edmundo G. 9004 Glenn Court Fairfax VA 22030	EM	OZBERKEM, MD, Vacit Y. 3301 Woodburn Rd, #307 Annandale, VA 22003	OBG	REARDON, MD, Wm John 6060 Arlington Blvd Falls Church VA 22044	GP *	SABELLA, MD, Lareine 6707 Old Dominion Dr McLean, VA 22101	OBG
MANLAPAZ, MD, Carolina Paredes 3014 W Cunningham Dr Alexandria VA 22309	FP	MORGAN, MD, Elizabeth 6862 Elm St, #420 McLean VA 22101	PS *	PALMERI, MD, Barbara Ann 301 W Maple Ave, #2-E Vienna VA 22180	P	RECINOS JR., MD, Adrian 6060 Arlington Blvd Falls Church VA 22044	PD	SACKS, MD, Charles B 1307 Vincent Place McLean, VA 22101	P
MANN, MD, Dean Le Mar Fairfax Hospital Emergency Rm Falls Church VA 22046	A *	MORTON, MD, Robert E 8301 Arlington Blvd, #4-5 Fairfax VA 22031	GE	PALOMBI, MD, Joseph John 1619 Hunting Avenue McLean, VA 22102	CHP *	RECIO, MD, Alfredo Hernandez 10720 Main Street Fairfax VA 22030	GP	SADR, MD, Manjeh 5202 Benton Avenue Bethesda, MD 20014	PD *
MARCUS, MD, Norman A. 8346 Traford Lane Springfield, VA 22152	ORS *	MOSS, MD, Morton Lionel 301 Maple Ave, W, #4-C-D Vienna VA 22180	DR	PAPPOUS, MD, Panagiotis 6807 Springfield Plaza #206 Springfield VA 22150	OTO *	REDLIN, MD, Wm Lloyd 3300 Gallows Rd, Falls Church VA 22046	DR *	SAGER, MD, Alan Robt 9604 Blincoe Ct Burke VA 22155	P
MARDER, MD, Carey Miles 133 Maple Ave, East Vienna, VA 22180	CD	MROCEK, MD, William J. 4600 King St, #4A Alexandria, VA 22302	CDS	PARK, MD, Tong Soo 3300 Gallows Rd, Falls Church, VA 22046	PD	REES, MD, Wm Chas 8988 Fern Park Dr Burke VA 22015	PD *	SAGER, MD, Dennis Wayne 1712 Club House Road Reston, VA 22090	IM
MARGULIES, MD, David M 8301 Arlington Blvd, #305 Fairfax VA 22031	OBG	MUELLER, MD, Karl H 6120 Brandon Ave Springfield VA 22150	P *	PASICOV, MD, Benjamin 7722 Crossover Drive McLean, VA 22101	EM	REILLY, MD, Michael Jos 1515 Old Chain Bridge Rd McLean VA 22101	IM *	SALAZAR, MD, Angel E 2024 Madrilion Road Vienna, VA 22180	NTR
MARION, MD, Edward David 6231 Leesburg Pike Falls Church VA 22044	OTO *	MULVANEY, MD, Richard Jos 1800 Old Meadow Rd, #706 McLean VA 22102	EM *	PASTORE, MD, Lucia 5522 Falmalead Road Fairfax, VA 22032	PTH	REING, MD, C. Michael 8301 Arlington Blvd Fairfax, VA 22030	ORS *	SALAZAR, MD, Delin B 3703 Mill Bank Ct Fairfax VA 22031	AN
MARTEL JR., MD, Leon Alphonse 2946 Sleepy Hollow Rd Falls Church VA 22044	OBG *	MURATORIO, MD, Jose Luis 8303 Arlington Blvd, #205 Fairfax, VA 22031	P	PAYNE, MD, Fred J 2945 Fort Lee St Herndon, VA 22071	GPM *	RENFELD, MD, Marilyn Lewis 2200 Leeland Drive Falls Church, VA 22043	CHP	SALIH, MD, Hassan A 3301 Woodburn Rd, #203 Annandale, VA 22003	CHP
MARTIN, MD, Dean H 1515 Chain Bridge Road McLean VA 22101	OBG *	MURIAS, MD, J.S. 6116 Rolling Road, Ste 104 Springfield VA 22152	PD	PEERBOOM, MD, Gerrit 3301 Woodburn Rd, #110 Annandale, VA 22003	GS *	REYNOLDS JR., MD, Arthur M 1515 Chain Bridge Rd McLean VA 22101	OPH *	SALUS, MD, Sydney Gordon 8360 Greensboro Dr, #501 McLean VA 22102	P *
MARTIN, MD, Diana 11200 A Lee Highway Fairfax, VA 22030	FP	MUROW, MD, Raymond J 6807 Springfield Pl, #201 Springfield VA 22150	IM	PEERBOOM, MD, Maud 9206 Coronado Terrace Fairfax, VA 22031	AN	REYNOLDS, MD, Brian Joe 1446 Laurel Hill Road Vienna VA 22180	GP	SALZBERG, MD, Allan M 8134 Old Keene Mill Road Springfield, VA 22152	AM
MARTIN, MD, John Oliver 2002 Friendship Lane Falls Church VA 22043	GP	MYERS, MD, Boyd Douglas 6400 Arlington Blvd, #330 Falls Church VA 22042	OPH *	PETERSON, MD, John Emerick 8316 Arlington Blvd Fairfax VA 22031	GS	RHYMERS, MD, Kurt Lee 5606 Wood Thrush Court Fairfax, VA 22032	OBG *	SANDERS, MD, John 313 Park Avenue Falls Church, VA 22046	OBG *
MASTERSON, MD, James H 611 S Carlin Sprgs Rd, #511 Alexandria VA 22204	ORS	NAM, MD, Jae Joong 7703 Killebrew Rd Annandale, VA 22003	OTO *	PFEFFER, MD, Bruce Wm 8316 Traford Lane Springfield VA 22152	PD	RIBEIRO, MD, Gilbert 3300 Gallows Road Falls Church, VA 22046	AN	SANTOS, MD, Rolando Jingco 8344 Traford Lane Springfield VA 22152	PUD *
MASTROTA, MD, Francis M 3709 George Mason Dr, #1605 Falls Church VA 22041	PD	NEDELCOVYCH, MD, Sava M 4600 King Street #5P Alexandria, VA 22302	GYN	PIESLOR, MD, Peter C. 8209 Guinevere Drive Annandale, VA 22003	NM *	RICH III, MD, William L 6231 Leesburg Pike Falls Church, VA 22044	OPH *	SCHACHNER, MD, Stephen Harold 6305 Castle Pl Falls Church VA 22044	IM *

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Michael J. Olichney, M.D.  
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Donald E. Bley, M.D.  
J. Thomas Ryan, M.D.  
Joseph D. Paquette, M.D.  
Paul W. Brammer, M.D.  
Nurse Practitioner,  
Patricia Sutherland

## **GASTROENTEROLOGY**

John C. Spivey, Jr., M.D.  
David B. Rice, M.D.

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Frank J. Durcan, M.D.

## **HEMATOLOGY/ONCOLOGY**

LeRoy J. Essig, M.D.

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Michael J. Olichney, M.D.  
Jerry A. Trice, M.D.  
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John C. Spivey, Jr., M.D.  
LeRoy J. Essig, M.D.  
Robert B. Vranian, M.D.  
Thomas E. Wheeler, M.D.  
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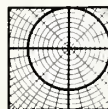


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		Norfolk VA 23505		Norfolk VA 23505		Norfolk VA 23510		Norfolk VA 23502	
		BOWERS, MD, John T	PM	CHIAVARINI, MD, Robt Louis	DR	ESCALANTE, MD, Guido Roger	FP	GOLDBERG JR., MD, Marvin	NEP
		850 Kempville Rd		3843 Thaxton Ln		9229 Granby St		6219 E Virginia Beach Blvd	
		Norfolk, VA 23502		Virginia Beach VA 23452		Norfolk VA 23503		Norfolk VA 23502	
		BOYD, MD, James Horace	GP	CHILDRESS JR., MD, A Jack	OBG	ESPEJO JR., MD, Guillermo	CD	GOLDMAN, MD, Chas Jay	IM
		549 E Brambleton Ave		512 S Independence Blvd		308 De Paul Med Bldg		935 Redgate Ave	
		Norfolk VA 23510		Virginia Beach, VA 23452		Norfolk VA 23505		Norfolk VA 23507	
		BOYD, MD, Wm Everett	PD	CHRISTIAN, MD, George Henry	DR	ESPINOSA, MD, Myrna Mendiola	US	GOODWIN, MD, Ambler Rey	PTH
		844 Kempville Rd Ste 204		3912 Coverdale Circle		1053 Birnam Woods Dr		De Paul Hosp	
		Norfolk VA 23502		Virginia Beach VA 23452		Virginia Beach VA 23464		Norfolk VA 23505	
		BREWER, MD, Herbert Martin	CD	CICCOONE, MD, Alvin Jacob	FP	ESTEP, MD, Herschel Leonard	IM	GOUGH, MD, Wm Wood	RHU
		205 Medical Tower		5205 Colley Ave		150 Kingsley Ln, De Paul Hosp		850 Kempville Road	
		Norfolk VA 23507		Norfolk VA 23508		Norfolk VA 23505		Norfolk VA 23502	
		BRICKMAN, MD, Robt David	CDS	CLAPP JR., MD, Henry W	AN	ETHERIDGE JR., MD, James E	PD	GOULD, MD, Randolph J	GS
		400 W Brambleton Ave, #200		503 Medical Tower		855 W Brambleton Ave		7510 Granby St	
		Norfolk VA 23510		Norfolk VA 23507		Norfolk VA 23510		Norfolk VA 23505	
		BRIDGES, MD, David Marvin	DR	COBBS, MD, Wilson N	U	EVETT, MD, Russell Dougherty	IM	GOULDIN, MD, Thos Winston	FP
		Norfolk General Hospital		549 E Brambleton Ave		530 Wainwright Bldg		9615 Granby St	
		600 Gresham Dr		Norfolk VA 23510		Norfolk VA 23510		Norfolk VA 23503	
		BOYD, MD, Wm Everett	PD	COFER JR., MD, Vernon L	IM	FAULCONER, MD, Robt Jamieson	PTH	GRAVES, MD, Chas Coakley	OS
		844 Kempville Rd Ste 204		5326 Edgewater Drive		201 Med Tower Bldg		2452 Jasper Court	
		Norfolk VA 23502		Norfolk VA 23508		Norfolk VA 23507		Norfolk VA 23518	
		BROCK, MD, Macon Foscoe	US	COOLEY, MD, Carl Conrad	OPH	FAULKNER, MD, Donald T	A	GREENSPAN, MD, Mark	GS
		6143 Rolfe Avenue		7320 Glenoie Ave, Apt 8C		901 Hampton Blvd		802 Medical Tower	
		Norfolk VA 23508		Norfolk VA 23505		Norfolk VA 23507		Norfolk VA 23507	
		BROOKS JR., MD, John	IM	CRISLER JR., MD, Crile	CDS	FAYTON, MD, Charles E	PD	GREGORY, MD, Roger Thorpe	CDS
		549 E. Brambleton Ave		400 W Brambleton Ave, #200		1732 Pope Street		250 W Brambleton Ave, #101	
		Norfolk, VA 23510		Norfolk VA 23507		Virginia Beach, VA 23464		Norfolk VA 23510	
		BROWN, MD, Ronald Bayard	EM	CROCKFORD, MD, Jon Lee	OBG	FEKETTE, MD, Andrew Maurice	IM	GRIFFEY, MD, Richard Thos	OPH
		2415 Spinnaker Court		903 Medical Tower		7930 F Chesapeake Blvd		801 Medical Tower	
		Virginia Beach VA 23451		Norfolk VA 23507		Norfolk VA 23518		Norfolk VA 23507	
		BROWNLEY, MD, Edwin T	IM	CROSS, MD, James Parker	OT	FELDMAN, MD, Frances R	P	GRINNAN JR., MD, R Bryan	IM
		555 Fenchurch Street		930 Redgate Ave		220 West Tazewells Way		5216 Edgewater Dr	
		Norfolk VA 23510		Norfolk VA 23507		Williamsburg, VA 23185		Norfolk VA 23508	
		BULLABOY, MD, Charles A	PDC	CROUCH JR., MD, Earl Russell	OPH	FERGUSON, MD, Chas Lee	OS	GRINNAN, MD, Geo Lamb Buist	CDS
		800 West Olney Road		2109 Windward Shore Dr		2409 Broad Bay Rd		400 W Brambleton Ave Ste 200	
		Norfolk VA 23507		Virginia Beach VA 23451		Virginia Beach VA 23451		Norfolk VA 23510	
		BURGER, MD, Robt Lindsay	IM	CULLEN JR., MD, Richard L	IM	FINCH, MD, Albert B	PD	GROSS, MD, Jerome S	GP
		250 W Brambleton Ave		401 Medical Tower		880 Kempville Rd, #2600		710 Liberty St	
		Norfolk VA 23507		Norfolk VA 23507		Norfolk VA 23502		Chesapeake VA 23324	
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		850 Kempville Road		1904 Claremont Ave		De Paul Med Bldg		68 Downtown Plaza	
		Norfolk, VA 23502		Norfolk VA 23507		Norfolk VA 23505		Norfolk VA 23510	
		BURKE, MD, Melvin H	AN	D'AMATO, MD, Nicholas Anthony	CLP	FINK, MD, Robert Alan	PD	GWATHMEY, MD, Frank W	HS
		211 De Paul Med Bldg		De Paul Hospital		410 Depaul Med Bldg		400 Gresham Dr	
		Norfolk VA 23505		150 Kingsley Lane		Norfolk, VA 23505		Norfolk VA 23507	
		BYRD, MD, John Abbott	IM	DAVIS JR., MD, Chas Emmett	GS	FITCHETT, MD, Claiborne W	GS	HARKINS, MD, Geo Archer	PDS
		809 Med Tower		810 Med Tower		844 Kempville Rd Ste 105		400 W Brambleton Ave Ste 200	
		Norfolk VA 23507		Norfolk VA 23507		Norfolk VA 23502		Norfolk VA 23510	
		BYRD, MD, Wm Edward	GYN						
		De Paul Med Bldg							
		Norfolk VA 23505							

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HASKELL JR, MD, Edward G E V M S, P O Box 1980 Norfolk VA 23501	FP	JEAN-GILLES, MD, Brunet 809 Medical Tower Norfolk VA 23507	GS	LANGLEY, MD, Beryl Cecilia E 1353 Dunstan Lane Virginia Beach VA 23455	CHP	MAY, MD, William H 5121 Greenview Rd Virginia Beach VA 23462	PD	NICHOLLS, MD, Richard B 901 Hampton Blvd Norfolk VA 23507	GYN
HAYES, MD, Henry Desmond 130 Colley Avenue Norfolk VA 23510	FP	JEFFERIES, MD, Allan H 312 De Paul Med Bldg Norfolk VA 23505	PD	LAREMONT, MD, Sylvia B. 1401 Tidewater Drive Norfolk VA 23504	OBG	MAYER, MD, Andrew Anthony Norfolk Gen Hosp, 600 Gresham Norfolk VA 23507	R	NOLD, MD, Ralph John 4700 Pruden Blvd Suffolk VA 23434	R
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HEIDE, MD, Robert Kay 5115 Studeley Ave Norfolk VA 23508	IM	JEROY, MD, Harry Keirn 4327 Thoroughgood Drive Virginia Beach VA 23455	AN	LEVIN, MD, Garshon J 815 Colonial Ave Norfolk VA 23507	GYN	MC COY, MD, Cullen M 7218 Shirland Ave Norfolk VA 23505	OPH	OLERICH, MD, Wm Lyle 844 Kempville Rd, #100 Norfolk VA 23502	CD
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HESS, MD, John Milton 503 Medical Tower Norfolk VA 23507	AN	JONES, MD, Georgaanna Seegar 603 Medical Tower Norfolk VA 23507	GYN	LIEN, MD, Buu That 8152 Jerrylee Drive Norfolk VA 23518	PD	MEREDITH II, MD, George Minor 844 Kempville Rd Ste 206 Norfolk VA 23502	OTO	PARISER, MD, Robert Jay 406 Medical Tower Norfolk VA 23507	D
HIGGINS, MD, Elizabeth M 600 Gresham Drive Norfolk VA 23507	ON	JORDAN, MD, Louis R 844 Kempville Rd, #101 Norfolk VA 23502	ORS	LILLY, MD, Edward Lewis 530 Wainwright Bldg Norfolk VA 23510	GE	MEREDITH JR, MD, H Clarkson 915 Hampton Blvd Norfolk VA 23507	IM	PARKER, MD, John Patrick 850 Kempville Road Norfolk VA 23502	CD
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HOFFLER JR, MD, William M 549 E Brambleton Avenue Norfolk VA 23510	IM	KERPELMAN, MD, Earle Jeroma 1000 Sunset Dr Norfolk VA 23503	GP	LITTLEPAGE, MD, Eleanor G M 941 Baldwin Avenue #B-1 Norfolk VA 23507	GYN	MILLER, MD, Jonathan W. 850 Kempville Road Norfolk VA 23502	IM	PAYNE, MD, William Duncan 209 De Paul Med Bldg Norfolk VA 23505	GS
HOFFLER, MD, Oswald W 549 E Brambleton Ave Norfolk VA 23510	GS	KESLER, MD, Robt Milton Norfolk Gen Hosp, 600 Gresham Norfolk VA 23507	R	LOVE, MD, Carolyn A 221 W Bute St Norfolk VA 23510	IM	MILLER, MD, Maurice M 207 Med Tower Norfolk VA 23507	GS	PEARLMAN, MD, Edwin 7312 Granby St Norfolk VA 23505	OPH
HOFFMAN, MD, George Charol 844 Kempville Rd Ste 105 Norfolk VA 23502	GS	KESSLER, MD, Charles A. 530 Wainwright Bldg Norfolk VA 23510	IM	LOVELL JR, MD, Charles F 2142 Haverford Dr Chesapeake VA 23320	PH	MILLER, MD, Newton Byrd 202 Depaul Med Bldg Norfolk VA 23505	OBG	PEEPLES, MD, William J Norfolk Gen Hosp, 600 Gresham Norfolk VA 23507	TR
HOOPER, MD, Wm 7304 Colony Pt Rd Norfolk VA 23505	GS	KIGHT, MD, John Randolph 4308 Sandy Bay Drive Virginia Beach VA 23455	GYN	LOWE, MD, Scott Miller 555 Fanchurch Street Norfolk VA 23510	OBG	MILLER, MD, Richard H 520 Glenegale Dr Virginia Beach VA 23462	R	PENIX, MD, Jerry O Don 607 Medical Tower Norfolk VA 23507	NS
HOPKINS JR, MD, John David 2539 Corpew Avenue Norfolk VA 23502	NM	KING, MD, John Norman 511 Medical Tower Norfolk VA 23507	GS	LOWELL, MD, William G. 1025 N George Wash Hwy Chesapeake, VA 23323	PD	MILLER, MD, Scott Arnold 850 Kempville Rd Norfolk VA 23502	IM	PERLMAN, MD, Jaroma David 1807 Whitle Lane Norfolk VA 23518	FP
HORDEN, MD, Harold Milton De Paul Medical Bldg, #408 Norfolk VA 23505	FP	KING, MD, John Winston 549 E Brambleton Ave Norfolk VA 23510	GS	LUNA, MD, Federico Martin 100 Marcy St Norfolk VA 23505	CD	MISTRY, MD, Zarine 106 Gary Player Road Portsmouth, VA 23701	IM	PHILIPPAKIS, MD, Spyros 203 Medical Tower Norfolk VA 23507	GS
HORTON, MD, Chas Edwin 400 W Brambleton Ave, #300 Norfolk VA 23510	PS	KING, MD, M Kirwan 914 Graydon Avenue Norfolk VA 23507	GS	MACHAJ, MD, Vincent R. 948 Royal Oak Close Virginia Beach VA 23452	AN	MOORE, MD, Alfred Andrew D 609 Colley Ave Norfolk VA 23507	CD	PICOU, MD, Wally John 605 Sarah Court Virginia Beach VA 23464	EM
HOSSAIN, MD, Mohammad A 3403 County Street Portsmouth, VA 23707	EM	KITTERMAN, MD, James S 1105 North Shore Road Norfolk VA 23505	GP	MAGEE JR, MD, William P 400 W Brambleton, #300 Norfolk VA 23510	PS	MORENO, MD, Leopold S D 7927 Old Ocean View Rd Norfolk VA 23518	FP	PLUNKETT JR, MD, Harry G 4301 Alifrinds Trail Virginia Beach VA 23455	FP
HOTCHKISS, MD, Wm S 2147 Old Greenbrier Rd Chesapeake, VA 23320	TS	KLOTZ JR, MD, Jeremiah A 221 Wainwright Bldg Norfolk VA 23510	OPH	MAGNESS II, MD, Alfred P. 935 Redgate Avenue Norfolk VA 23507	NS	MORGAN JR, MD, Franklin G. 844 Kempville Rd, #208 Norfolk VA 23502	OBG	POLE, MD, William C 844 Kempville Rd, #101 Norfolk VA 23502	ORS
HOVLAND, MD, William Neal De Paul Medical Bldg, #309 Norfolk VA 23505	IM	KNIGHT JR, MD, Morris Reed 1900 Lynn Cove Lane Virginia Beach VA 23454	R	MAHAN, MD, Jack Delano 600 Gresham Dr Norfolk VA 23507	R	MORTON, MD, Robt A 915 Hampton Blvd Norfolk VA 23507	IM	PORETTA, MD, Jerome C 503 Medical Tower Norfolk VA 23507	AN
HUBBARD II, MD, George W 844 Kempville Rd, #105 Norfolk VA 23502	GS	KOEHL, MD, Geo Wm 330 Brambleton Ave Norfolk VA 23510	PD	MANICKAVASAGAR, MD, Marie J 409 Maiden Lane Chesapeake VA 23325	NEP	MOSQUERA, MD, Guillermo De Paul Medical Bldg, #512 Norfolk VA 23505	U	PORTER JR, MD, Frederick S 800 West Olney Road Norfolk VA 23507	PD
HUMPHREY, MD, William Trowell 905 Redgate Avenue Norfolk VA 23507	OPH	KREGER, MD, David Lawrence 400 W Brambleton Ave, #103 Norfolk VA 23510	GE	MANICKAVASAGAR, MD, S. 409 Maiden Lane Georgetown Pt Chesapeake VA 23325	GS	MOSS, MD, Burton Alan 112 E Little Creek Rd Norfolk VA 23505	AI	PORTER, MD, Ira Stanley 6275 E Va Beach Blvd, #300 Norfolk VA 23502	ORS
HURWITZ, MD, Richard L 6219 Va Beach Blvd, #3 Norfolk VA 23502	CDS	KRISCHER, MD, Meyer I 6027 River Rd Norfolk VA 23505	LM	MANN, MD, Robt Fletcher 902 Graydon Avenue Norfolk VA 23507	IM	MUHLENDORF, MD, Ivan K 844 Kempville Rd #208 Tidewater Phys For Women Norfolk VA 23502	OBG	POSSNER, MD, Irvin L. 210 De Paul Med Bldg Norfolk VA 23505	OPH
IBARRA, MD, Jorge 210 Southern Off Bldg Norfolk VA 23505	GS	KRUGER, MD, David B 511 N. Shore Rd. Norfolk VA 23462	P	MARESH, MD, Chas Geo 309 De Paul Med Bldg Norfolk VA 23505	IM	MULLEN, MD, Jos Terrance 7339 Ruthven Road Norfolk VA 23505	GS	POWELL JR, MD, Albert Henry 20 Koger Executive Center 212 Norfolk VA 23502	P
ISAAC, MD, Joseph William 116 Brentwood Circle Virginia Beach VA 23452	OBG	KRUGER, MD, Howard I 1723 Cromwell Rd Norfolk VA 23509	FP	MARKHAM, MD, Thomas Carl 844 Kempville Rd, #101 Norfolk VA 23502	ORS	MURPHY JR, MD, Wm F 330 W Brambleton Hague Tower Norfolk VA 23510	PD	PRICE, MD, James Dalton 6833 Gardner Dr Norfolk VA 23518	IM
IVES, MD, Charles Everett De Paul Medical Bldg, #209 Norfolk VA 23505	GS	KUMAR, MD, Achla De Paul Medical Bldg, #305 Norfolk VA 23505	PM	MAROTO JR, MD, Felix 503 Medical Tower Norfolk VA 23507	AN	MURRAY, MD, Kevin Patrick 142 W York St Norfolk VA 23507	IM	PROPERT, MD, David B. E V M S, Box 1980 Norfolk VA 23501	IM
JACKSON, MD, Robert T. 901 Hampton Blvd Norfolk VA 23507	OTO	LADAGA, MD, Leopoldo Elio 4713 Five Forks Court Virginia Beach VA 23455	PTH	MARTENS JR, MD, Werner 902 Graydon Ave Norfolk VA 23507	IM	NAYLOR, MD, Wm Talbott 902 Graydon Avenue Norfolk VA 23507	IM	PRYOR, MD, Donald Chas 1629 Arrowhead Rd Virginia Beach VA 23455	EM
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REDA, MD, Annette Williams 801 Medical Tower Norfolk VA 23507	OPH	SHAPIRO, MD, Saml Leon 844 Kempville Rd #100 Norfolk VA 23502	N	TAYLOR, MD, Jack Borden De Paul Hospital Norfolk VA 23505	CD	WILLARD, MD, Richard Norman 4600 Southern Pines Dr Virginia Beach VA 23462	OBG	MC INTYRE, MD, William Wallace Internal Medicine Group Nassawadox VA 23413	GE
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REINA, MD, Abdon De Paul Medical Bldg, #302 Norfolk VA 23505	NS	SHELTON, MD, Jean Elizabeth 601 Jones Street Sulfolk VA 23434	PD	TERZIS, MD, Julia K. 400 W Brambleton Ave, #300 Norfolk VA 23510	PS	WILLIAMSON, MD, Sterling R. 6275 E Va Beach Blvd, #300 Norfolk VA 23502	ORS	MOLERA, MD, Federico F. P O Box 819 Nassawadox, VA 23413	AN
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RESHEFSKY, MD, Bonnie Louis De Paul Med Bldg Ste 301 Norfolk VA 23505	OPH	SIMPSON, MD, Geo Winslow 3808 E Indian River Rd Chesapeake, VA 23325	GP	THOMAS, MD, P. Varkey P O Box 9170 Norfolk VA 23505	AN	WILSON, MD, Robt Marion 6219 E. Virginia Beach Blvd Norfolk, VA 23502	NEP	PAYNE II, MD, Philip M Nassawadox, VA 23413	U
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<b>PATRICK HENRY</b>									
ADAMS, MD, Saml Webster 9 Hickory Ridge Rd Martinsville VA 24112	FP	PRICE, MD, Homer H Hospital Dr Martinsville VA 24112	GP	CHAUDHURI, MD, Mohan Lal Dominion Natl Bank, #210 Portsmouth, VA 23704	OTO	HOLLIS, MD, Joseph B 1211 Rodman Ave Portsmouth, VA 23707	GE	OSTROFF, MD, Edwrd Benj 4037 Taylor Rd, #A Chesapeake, VA 23321	U
ALBANESE, MD, Robert 1005 Cherokee Trail Martinsville VA 24112	EM	PRINCE III, MD, William D. 314 Fairy Street Martinsville, VA 24112	IM	CHEN, MD, Chun-Ming 3315 County Street Portsmouth VA 23707	AN	HOLLOWELL, MD, John W 226 Fort Lane Portsmouth VA 23704	U	PADGETT, MD, Thos E 16 Shennock Dr Portsmouth VA 23701	R
BERGMAN, MD, Stuart M 15 Cleveland Ave Martinsville, VA 24112	U	RANDALL, MD, Eugene H. P O Box 4243 Martinsville, VA 24115	P	CHEHAULT JR, MD, Oran Ward 4037 Taylor Road, #A Chesapeake, VA 23321	U	HOLMES, MD, Francis Hammond P O Box 6658 Portsmouth VA 23703	R	PARK, MD, Crawford Dick Olde Towne Medical Center Portsmouth VA 23704	CDS
BESTLER, MD, J Michael Med Center Ste 204 Hosp Dr Martinsville VA 24112	PS	RICHMAN, MD, Donald Wm Box 3151 Martinsville VA 24112	OPH	CHOUDHURY, MD, Ali Azem 711 Rodman Avenue Portsmouth VA 23707	NEP	HOOD, MD, Kaseidul 3116-A Tyre Neck Road Portsmouth VA 23703	FP	PARK, MD, Philip Merlin 4057 Taylor Road Chesapeake, VA 23321	OBG
CAMPBELL, MD, Jos Cameron Medical Center, Box 4546 Martinsville VA 24112	R	RICHMOND, MD, Marion D P O Box 5351 Martinsville VA 24112	PD	CHOUGH, MD, Dee Been 4041 Taylor Rd, #G Chesapeake, VA 23321	CD	ISLAM, MD, Mohammed Aminul 3736 Western Branch Blvd Portsmouth VA 23701	IM	PASCUAL, MD, Reymond G. 622 Citizens Trust Bldg Portsmouth VA 23704	GS
CAMPBELL, MD, Henry S. Box 3151 Martinsville VA 24115	OPH	RIDER, MD, Robt Edward Box 5351 Martinsville, VA 24112	PD	CHOY, MD, Yoon Keun 3419 King Street Portsmouth, VA 23707	AN	JACKSON, MD, John A. 3329 Deep Creek Boulevard Portsmouth VA 23702	GP	PASCUAL, MD, Vienmer G 968 S. Spiegel Drive Virginia Beach VA 23454	EM
CHADDOCK, MD, William Moran 4301 West Markham Little Rock, AR 72205	NS	ROBBINS JR, MD, Wm Lacy Box 4111 Martinsville VA 24112	GP	CLARE, MD, Frank B 3500 Queen St Portsmouth VA 23707	NS	JAMES, MD, Hillman Holmes Pess House Cretford At London Portsmouth VA 23704	P	PERWAIZ, MD, Jevaid A 3003 Churchland Blvd Chesapeake VA 23321	OBG
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EBERHART, MD, Jack Henry 435 Commonwealth Blvd Martinsville, VA 24112	OTO	SHERMAN, MD, Claude Porter P O Box 3723 Martinsville VA 24112	ORS	CRETEUR, MD, Christian E 3419 King St Portsmouth VA 23707	AN	KNAPP, MD, Robt Woodruff Dept Ot Surgery, Vamc Hampton, VA 23667	GS	PRICE, MD, Neil D. 100 Pinewood Road, #226 Virginia Beach, VA 23451	P
ENGEL, MD, John Jos P O Box 5351 Martinsville VA 24112	PD	SMITH, MD, John Randolph 314 Fairy Street Martinsville VA 24112	IM	DAJAO, MD, Rise Faith 1105 Halitax Avenue Portsmouth VA 23707	GP	KNAUFT, MD, Richard David 3300 High Street Portsmouth VA 23707	ORS	PSIMAS, MD, George N 2929 London Blvd Portsmouth VA 23707	ORS
FINCH, MD, Robt Delmar 207 Woodlawn Rd Collinsville VA 24078	R	SZULECKI, MD, Judith Marie 15 Cleveland Avenue Martinsville VA 24112	D	DARBY, MD, Daniel Lee 209 62nd Street Virginia Beach, VA 23451	P	KOSTINAS, MD, John E 4037 Taylor Road Chesapeake VA 23321	HEM	PYLE JR, DO, Wellden 3116 A Tyre Neck Rd Portsmouth VA 23703	OST
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FRENCH, MD, John D P O Box 5351 Martinsville VA 24115	PD	WALLACE, MD, George Lamar 1100 Spruce Street Martinsville, VA 24112	IM	DAYANIM, MD, Behrooz 620 London Blvd, #300 Portsmouth VA 23704	GS	KUO, MD, Hwang Ren 618 Geo Washington Hwy N Chesapeake VA 23323	FP	ROBERTSON, MD, Wm Clayton 416 Citizens Trust Bldg Portsmouth VA 23704	OPH
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HOLSINGER, MD, Donald Rider 15 Cleveland Ave Martinsville VA 24112	IM	<b>PORTSMOUTH</b>		DENARO JR, MD, Frank 3315 County St Portsmouth VA 23707	OBG	LENTZ, MD, Edmund T P O Box 103 Greentown, PA 18426	OM	ROSEN, MD, David Aaron Crawford At London Portsmouth VA 23704	P
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IRBY, MD, Jethro Hurt Hospital Drive Martinsville VA 24112	GP	ATLAS, MD, Barry Foster 3603 County Street Portsmouth VA 23707	OPH	DOMMISSE JR, MD, John 1801 Airline Blvd Portsmouth VA 23707	P	LIPMAN, MD, Ansel 520 Peninsula Ave Portsmouth VA 23704	R	SAN DIEGO, MD, Carmelite M Portsmouth Gen Hosp Dept Path Portsmouth VA 23704	CLP
JESNECK, MD, Edward Route 1, Box 147 Bassett VA 24055	FP	BAKER, MD, Robt E 3603 County St Portsmouth VA 23707	OPH	DOZORETZ, MD, Ronald Irving Crawford Parkway At Fort Lane Portsmouth VA 23704	P	LOEW, MD, Albert G. 3500 Queen Street Portsmouth, VA 23707	NS	SARKAR, MD, Dilip Kumar 618 Citizens Trust Bldg Portsmouth VA 23704	GS
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		BARRECA JR, MD, Joseph Peter 3500 South St Portsmouth VA 23707	OS	FERGUSON, MD, William P O Box 6031 Suffolk VA 23433	US	MANCUSO, MD, Frank Smith 3620 Clifford St Portsmouth VA 23707	PD	SAYEGH, MD, Emile Selim 226 Fort Lane Portsmouth VA 23704	U

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SCHLANGER, MD, Maurice R 601 Rodman Ave Portsmouth VA 23707	IM	PRINCE WILLIAM	ABELA, MD, Augusto V 14316 Jefferson Davis Hwy Woodbridge, VA 22191	OPH	HERITAGE, MD, Douglas E 2026-A Opitz Blvd Woodbridge, VA 22191	OBG	PARKER III, MD, Frederick W 8620 Rolling Road Manassas, VA 22110	U	ZAZZARO, MD, Patrick Francis 9703 Sudley Manor Drive Manassas, VA 22110	DR	
SCHWEIGER JR., MD, Ernst 2486 Airline Blvd Portsmouth VA 23701	IM	ALISUAG JR., MD, Andres 8703 Stonewall Rd, #33 Manassas, VA 22110	IM	IRWIN, MD, Gilbert Raymond 9590 Surveyor Court Manassas VA 22110	IM	JARBADAN, MD, Ignaz Papa 1966 Opitz Boulevard Woodbridge VA 22191	IM	PAUSWINSKI, MD, John Robt 9613 Champion Court Manassas VA 22110	FP	RICHMOND	
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ST GEORGE, MD, John R 307 Park Road Portsmouth VA 23707	GP	DESPER, MD, Paul Carlton 9608 Champion Court Manassas VA 22110	IM	FERLAZZO, MD, Stephen L P O Box 442 Woodbridge VA 22194	OTO	FINEMAN, MD, Bill L 3126 Davis Ford Road Woodbridge, VA 22191	GER	FRANCIS, MD, Jamison K 14904 Jeff Davis Hwy Woodbridge, VA 22191	D	AGATE, MD, Gao H 6 A P Hill Avenue Highland Springs VA 23075	PH
STENICKA, MD, Francis John 3800 Poplar Hill Rd, #D Chesapeake, VA 23321	EM	FRANKEL, MD, Nicholas P P O Box 1847 Alexandria, VA 22313	DR	GARVEZ, MD, Magdalena D 2321 Princess Anne Lane Woodbridge VA 22191	OBG	GILMONTERO, MD, Guillermo H 9580 Surveyor Court Manassas VA 22110	ORS	GLUCK, MD, Gabriel 8702 Sudley Road Manassas VA 22110	ORS	AGHDAMI, MD, Aliasghar 2831 Newquay Lane Richmond VA 23236	AN
SU, MD, Robert K 2905 Tanbark Lane Portsmouth VA 23703	AN	GOWER, MD, Arthur Gaillard 8722 Sudley Rd Manassas VA 22110	PD	GUERRERA, MD, Chas Jos 8717 Digges Rd Manassas VA 22110	OBG	GUERRERO, MD, Victor N 9036 Sudley Rd Manassas VA 22110	ORS	GUIEB, MD, Adelaida 9407 Lafayette Ave Manassas VA 22110	GP	AL-ABDULLA, MD, Hamid 3604 Monument Ave Richmond VA 23230	CD
TANAKA JR., MD, Zenji 3419 King St Portsmouth VA 23707	AN	HAHN, MD, Gunter Ernst 8708 Sudley Road Manassas VA 22110	IM							ALAVI, MD, S. Manucher 1602 Skipwith Rd Richmond VA 23229	R
TENEKJIAN, MD, Vasken Kevork 620 London Blvd Portsmouth, VA 23704	TS									ALLEN JR., MD, Benj Randolph 1651 Parham Road Richmond VA 23229	NS
TERRY, MD, Wm Sanford 3300 High St Portsmouth VA 23707	IM									ALLEN, MD, Banj Randolph 7005 Marcliff Court Richmond VA 23228	GP
THANADAR, MD, Abu Abdur 422 Citizens Trust Bldg Portsmouth VA 23704	U									ALMOND, MD, Hilton Robinson 7702 Parham Road Richmond VA 23229	GE
THOMASON, MD, Phillip Ray 3217 Stamford Rd Portsmouth VA 23703	PD									ANDERSON, MD, Wm Morris 306 Tuckahoe Blvd Richmond VA 23226	IM
TIESSENGA, MD, Sidney W 110 American Legion Rd Chesapeake, VA 23321	ORS									ANDRAKO, MD, John David 6823 Kensington Avenue Richmond, VA 23226	PD
TRUE, MD, De Witt Sidney 4426 Point West Drive Portsmouth VA 23703	GYN									ANDREWS, MD, Jack Praston 1001 Hoaks Road Richmond VA 23225	PD
TURNER, MD, Franklin C 2800 Acres Rd Portsmouth VA 23707	IM									ANSELL, MD, Burnass Fardmand 7702 Parham Road Richmond VA 23229	IM
VALDIVIESO, MD, Jorge R 3800 Shoreline Dr Portsmouth VA 23703	OPH									ANTE, MD, Nilda R 2105 E. Parham Rd Richmond VA 23228	OBG
VIZCAINO, MD, Federico 355 Crawford Parkway, #514 Portsmouth, VA 23704	P									ANTHONY, MD, Scott Bryan Box 515, M C V Station Richmond, VA 23298	ORS
WARDELL, MD, Arthur W 2929 London Blvd Portsmouth, VA 23707	ORS									APPERSON, MD, Wm Eugene 905 Pine Ridge Rd Richmond VA 23226	PUD
WARNING, MD, Milton G 301 Fort Lane Portsmouth, VA 23704	CHP									ARCHER JR., MD, John Stanard 209 Med Arts Bldg Richmond VA 23219	OTO
WEBB, MD, Thos Harry 3300 High St Portsmouth VA 23707	IM									ARCHULETA, MD, Bobby Arnold 4757 Cochise Trail Richmond, VA 23234	PD
WEITZMAN, MD, Gerald 2929 London Blvd Portsmouth VA 23707	ORS									ARNOLD, MD, Gayle G Box 7348 Richmond VA 23221	PD
WHEELER, MD, William F P O Box 6658 Portsmouth, VA 23703	DR									ASHBY, MD, Franklyn Henry Box 26603 Richmond VA 23261	OM
WHITBECK, MD, John Volkert P O Box 6658 Portsmouth, VA 23703	DR									ASHWORTH, MD, John Shariden 3540 Floyd Ave Richmond VA 23221	IM
WILHITE JR., MD, Philip A 4723 Rivershore Rd Portsmouth VA 23703	D									ATYIEH, MD, Wasfi A 3500 Kensington Ave Richmond VA 23221	OTO
WILLIAMSON, MD, Neihl J. 601 Rodman Road Portsmouth, VA 23707	US									ATKINSON, MD, Garald Waslay 1415 Johnston Willis Dr Richmond VA 23235	N
WILSON, MD, Kevin D 3300 High St Portsmouth, VA 23707	IM									ATWILL, MD, Wm Henry 5224 Monument Ave Richmond VA 23226	U
WINSTON, MD, Wm O 3300 High St Portsmouth VA 23707	OBG									AUSTIN, MD, David H. 4865 Finlay Street Richmond, VA 23231	PD
WITT, MD, Frederick Jos 3108 Tyre Neck Rd Portsmouth VA 23703	OBG									AUSTIN, MD, Leonard Anthony 205 Oxford Circle East Richmond VA 23221	PD
WOLF, MD, Jeffrey Stephen 620 London Blvd Portsmouth, VA 23704	GS									AWAD, MD, Allen J 2103 E Parham Road Richmond VA 23228	OBG
WOLOY, MD, Eleanora Marie 4696 Honeygrove Rd, #104 Virginia Beach, VA 23455	CHP									AYRES, MD, John W 7135 Jahneke Rd Richmond VA 23225	ORS
WU, MD, Shue Chen 3701 South Street Portsmouth, VA 23707	OBG									BAGLEY, MD, John J. 9606 Patterson Ave Richmond, VA 23229	OBG
YARBROUGH, MD, Terry Pinckney 3300 High St Portsmouth VA 23707	IM									BALLIE, MD, Allston Gibbs 204 N Hamilton St Richmond VA 23221	PUD

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BAIN, MD, Francis Nott Box 669 Goochland, VA 23063	FP	BLANCHARD III, MD, Lawrence E 5600 Grove Avenue Richmond VA 23226	D	BROOCKER, MD, Warren Alan 7151 Jahanke Road Richmond VA 23225	OBG	CADY, MD, Allan Bartlett Rt 1 Box 422 Mechanicsville VA 23111	GE	CHRISTIE, MD, Laurence Glenn 7601 Forest Ave #115 Richmond VA 23229	GS
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BARR, MD, Wm Clayton 5507 Toddbery Rd Richmond VA 23226	R	BLANTON, MD, Erika M 901 Hoaks Road Richmond VA 23225	OBG	BROOKS, MD, Kenneth Phillip 7149 Jahanke Road Richmond VA 23225	P	CALKINS, MD, Ronald Fleming 5801 Brems Road Richmond VA 23226	R	CLARK, MD, Mark Wayne 700 West Grece St, #204 Richmond, VA 23220	CD
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BARRINGER, MD, Michel Laron Box 7348 Richmond VA 23221	PD	BLAYLOCK, MD, Wilmer Kenneth Box 164 Richmond VA 23298	D	BROWN III, MD, Alexander G 1615 Henover Avenue Richmond, Va 23220	IM	CALL, MD, John Danl 8921 Norwick Rd Richmond VA 23229	IM	CLARY, MD, Beverley B 4315 Grove Ave Richmond VA 23221	ORS
BASKERVILLE, MD, A. L. 700 W Grace St, #204 Richmond VA 23220	CD	BLOUNT, MD, Alston Wilcox 2002 Brems Rd Lower Level Richmond VA 23226	CD	BROWN JR., MD, Leon Junius 1923 Decatur Street Richmond VA 23224	FP	CALLOWAY, MD, Wm Christian 8 Esl Leigh St Richmond VA 23219	FP	CLARY, MD, Richard M. 425 North Boulevard Richmond VA 23220	GS
BATES 111, MD, Robley D. 1805 Monument Ave, Ste 511 Richmond VA 23220	U	BLUMBERG, MD, Michael Zengwill 2000 Brems Rd, #204 Richmond VA 23226	AI	BROWN, MD, Edwin Merriman Va St Hill Dept 109 Governor Richmond VA 23219	PH	CAMETAS, MD, John Gus Parham & 3 Chopl Rds, #1 Richmond VA 23229	FP	CLEMENT, MD, Stephen 7601 Forest Ave, #332 Richmond VA 23229	GE
BATES JR., MD, Hampton Robert 7101 Jahanke Road Richmond VA 23225	NM	BOARD, MD, Anne J Woodrum 1407 Cummings Dr A H Robins Co Richmond VA 23220	PA	BROWN, MD, James La Velle 2004 Brems Road Richmond VA 23226	OPH	CAMP, MD, Paul Douglas 7601 Forest Ave, Ste 228 Richmond VA 23229	CD	CLEMENTS JR., MD, Ernest L 7601 Forest Ave, Ste 228 Richmond VA 23229	ORS
BATTISTA JR., MD, Jos Victor 5855 Brems Rd Ste 205 Richmond VA 23226	OBG	BOARD, MD, John Arnold Box 34, M C V Station Richmond VA 23298	OBG	BROWN, MD, Peter Wilcox 417 Libbie Avenue Richmond VA 23226	GS	CAMPBELL, MD, James Ashlon 8325 Brookfield Road Richmond VA 23227	FP	COBAUGH, MD, Donn Stephen 10050 Dimrock Drive Richmond VA 23235	US
BAXTER, MD, Robt Wallace 1600 Conderate Ave Richmond VA 23227	GP	BOATWRIGHT III, MD, Joseph W. P O Box 26591 Richmond VA 23261	PD	BRUCH, MD, Wm Mark Box 506, M C V Station Richmond VA 23298	PD	CAMPBELL, MD, Rulh F Williams 7435 Riverside Dr Richmond VA 23225	PD	COHEN, MD, Irwin Kelman Box 154, M C V Station Richmond VA 23298	PS
BEACHLEY, MD, Michael Chas Box 2, M C V Station Richmond VA 23298	R	BOKINSKY, MD, Gary Brooks 5224 Monument Avenue Richmond VA 23226	U	BRUGH III, MD, Viclor Miller 10307 Meremont Dr Richmond VA 23233	EM	CANE, MD, James Howard P O Box 26786 Richmond VA 23261	OBG	COHEN, MD, Stephen Alen Box 34, M C V Station Richmond VA 23298	OBG
BEAZLEY III, MD, Wyatt S 425 North Boulevard Richmond VA 23220	GS	BONES, MD, Jos Thos 4906 Forest Hill Ave Richmond VA 23225	PD	BRUMMER, MD, Donelud Louis Box 204, M C V Station Richmond VA 23298	ID	CARAVATI JR., MD, Chas Merin 5600 Grove Ave Richmond VA 23226	D	COLE, MD, Deen Baldwin 30 Maxwell Rd Richmond VA 23226	IM
BECK, MD, Ralph Edmund 5801 Brems Rd Richmond VA 23226	PTH	BONNER, MD, Charles H. 1311 Palmyra Ave Richmond VA 23227	PM	BRUSH, MD, John J. 1805 Monument Ave Richmond VA 23220	N	CARAVATI, MD, Chas Martin 208 Gun Club Rd Richmond VA 23221	GE	COLE, MD, Waverly Manson 1308 Grove Ave Richmond VA 23220	AN
BECKER, MD, Donelud Paul Box 631 Richmond VA 23298	NS	BOON, MD, Franklin F 515 North 10th Street Richmond VA 23219	CHP	BYRCE II, MD, Edwin Clinton 5406 New Kent Rd Richmond VA 23225	OPH	CARDEA, MD, John A 13717 Hickory Nut Point Midlothian, VA 23113	ORS	COLEMAN, MD, Custis Lensing 5855 Brems Rd, Bldg 502 Richmond VA 23226	GS
BECKER, MD, Elmore James 1815 Ivy Stone Drive Richmond VA 23233	IM	BOONE JR., MD, Elwood Bernerd 1400 Westwood Ave, Ste 304 Richmond VA 23227	U	BYRCE JR., MD, Wm Fielding 8716 Brown Summit Rd Richmond VA 23235	FP	CARDWELL, MD, Chas Patteson 101 Tuckahoe Blvd Richmond VA 23226	AN	COLL, MD, Jose D 3628 S Belmont Rd Richmond VA 23234	GS
BECKER, MD, Stanley Harold 2807 Pennington Rd Richmond VA 23229	D	BOOTH, MD, Jerry Clark 112 Charnwood Rd Richmond VA 23229	D	BRYSON, MD, Gilbert Hamilton 7702 Parham Rd Richmond VA 23229	GS	CARMICHAEL, MD, Miriam W 3919 Seminary Ave Richmond VA 23227	N	COLLIER, MD, John E 1717 Bellevue Ave Richmond VA 23227	GP
BEDINGER SR., MD, Robert W 7702 Parham Road Richmond VA 23229	IM	BOSHER JR., MD, Lewis Hinton 103 Seneca Road Richmond VA 23226	TS	BUDJ JR., MD, Semi Walthell 5500 Monument Ave, Ste 1 Richmond VA 23226	IM	CARPENTER, MD, Earnesl B 4315 Grove Ave Richmond VA 23221	ORS	CONQUEST, MD, H. Feirlax 4 Calycanthus Road Richmond VA 23221	ORS
BELGRAD, MD, Richard 601 Heathfield Rd Richmond VA 23229	TR	BOSHER, MD, Linwood Paul 417 Libbie Avenue Richmond VA 23226	GS	BUIS, MD, L. James 8802 Sierra Rd Richmond VA 23229	PUD	CARRIGAN, MD, Edward P 2911 Grove Avenue Richmond VA 23221	ORS	CONSTANT JR., MD, Tony 1603 Lake Ave Richmond VA 23226	EM
BELLE, MD, Walton Mc Neil 2901 Brook Rd Richmond VA 23220	GS	BOWERS, MD, Russell V 3500 Mechanicsville Pk Richmond VA 23223	GP	BULLOCK JR., MD, Henry A. 2019 Monument Avenue Richmond VA 23220	OBG	CARTER II, MD, B Nolend 1401 Johnston Willis Dr Richmond VA 23235	GS	COOKE, MD, Charles Lee 7702 Parham Road Richmond VA 23229	RHU
BENNETT, MD, L P Robinson 2809 North Ave Richmond VA 23222	PD	BOWMAN, MD, John David 2911 Grove Avenue Richmond VA 23221	ORS	BULLOCK JR., MD, J. Paul 5855 Brems Rd, #508 Richmond VA 23226	OPH	CARTER JR., MD, Hill Box 958 Ashland VA 23005	FP	COOKE, MD, Semi L 917 W Murray Ave Durham NC 27704	OTO
BENNETT, MD, Robert M. P O Box 13470 Richmond VA 23225	CD	BOWSER, MD, Barrington H 1807 Hampton St Richmond VA 23220	PD	BULLOCK, MD, John Boyd 2019 Monument Ave Richmond VA 23220	IM	CARTER, MD, Wesley Byrd 3001 Fifth Avenue Richmond VA 23222	CHP	COOKSEY, MD, Wm Perry 5700 W Grace St Richmond VA 23226	OBG
BENTLEY, MD, Jack Kitchener 11540 Rexmoor Drive Richmond VA 23236	R	BOYAN, MD, Chas P 7 Calycanthus Lane Richmond VA 23221	AN	BUNDY 111, MD, Walter E. 5855 Brems Rd, Ste 508 Richmond VA 23226	OPH	CASPARI, MD, Richard B 199 Three Chopt Rd Richmond VA 23229	ORS	CORCORAN, MD, James F.T. 1500 Westbrook Ave Richmond VA 23227	P
BEORN, MD, Charles F. 2101 Carbon Hill Drive Midlothian, VA 23113	IM	BOYCE JR., MD, Edward L. Box 9046 Richmond VA 23225	FP	BUNDY JR., MD, Walter E. 6823 Kensington Ave Richmond VA 23226	PDA	CATLETT, MD, John B 7702 Parham Road Richmond VA 23229	A	COUK, MD, Mecon Smiley 3800 Patterson Ave Ste 101 Richmond VA 23221	IM
BERREY, MD, Bedford H 4431 Old Foxtrail Midlothian, VA 23113	PD	BOYCE, MD, Stanley Carlton 114 Homestead Drive Colonial Heights, VA 23834	GP	BURCH, MD, Chas Dick 5855 Brems Rd, #308 Richmond VA 23226	PD	CAVALCANTI, MD, Eduardo Jose 2701 Cherrytree Lane Richmond VA 23235	TR	COX, MD, Wm Henry 105 Virginia Ave Richmond VA 23226	OBG
BETTS, MD, Martin Frederick 7601 Forest Ave, Ste 222 Richmond VA 23229	PUD	BOYD, MD, Milton Alexander Parham Med Center Richmond VA 23229	IM	BURKE III, MD, George Wilson 1400 Westwood Avenue, #305 Richmond VA 23227	PUD	CHALKLEY, MD, Thos Spencer 1013 Westham Parkway Richmond VA 23229	PD	COXE III, MD, Jos Wentworth 7702 Parham Rd Richmond VA 23229	GS
BICK, MD, Michael S. 5855 Brems Rd, #G 4 Richmond VA 23226	P	BRADENHAM, MD, Ben Persons 7601 Forest Ave, Ste 332 Richmond VA 23229	IM	BURKE JR., MD, Arthur Wade 1104 West Franklin Street Richmond VA 23220	TR	CHAN, MD, James C M Box 498, M C V Station Richmond VA 23298	PD	CRAIGGS III, MD, Thomes F. 2247 Loch Braemar Dr Richmond VA 23236	FP
BICKERS, MD, Wm M 1802 Brooktree Court Richmond VA 23233	GYN	BRADLEY, MD, Robert W. Box 9 Powhatan VA 23139	GP	BURKE, MD, Petrick K. 413 Stuart Circle Richmond VA 23220	IM	CHAPIN, MD, Wm Evans Rt 1 Box 17 Callands VA 24530	PD	CRICHGNO, MD, G. A. 1805 Monument Ave, #314 Richmond VA 23220	CHP
BINFORD, MD, Charles A 7149 Jahanke Rd Richmond VA 23225	P	BREDRUP JR., MD, Ole C St Mary Hosp Depl R Richmond VA 23226	R	BURKHARDT, MD, Barry W. 1447 Johnston Willis Dr Richmond VA 23235	ORS	CHAPLIN JR., MD, Robt Rogers 7111 Jehnke Rd Richmond VA 23225	FP	CRITTENDEN, MD, David Grey 3536 Grove Ave Richmond VA 23221	OTO
BINHAMMER, MD, Harold E 5001 W Village Gr Dr, #209 Midlothian, VA 23113	FP	BREEDEN, MD, Louis M. 7702 Parham Road Richmond VA 23229	FP	BUTTERWORTH III, MD, John F 1447 Johnston-Willis Dr Richmond VA 23235	ORS	CHARITY, MD, Cynthia M. P O Box 5384 Richmond VA 23220	PD	CROOKS JR., MD, Lewis Denl 3802 Old Gun Road West Midlothian VA 23113	OBG
BIRO, MD, Victor G 2201 Westwood Ave Richmond VA 23230	HEM	BREHMER, MD, Chas Edward 731 Milbrae Rd Richmond VA 23236	GP	BUTTERWORTH, MD, Thomas R 2500 Pocoshock Pl Richmond VA 23235	ORS	CHARITY, MD, Renard Adkins P O Box 5384 Richmond VA 23220	OBG	CROSSEN, MD, Richard Wm 2301 Hilliard Road Richmond VA 23228	FP
BLACK JR., MD, James B 1833 Monument Ave Richmond VA 23220	IM	BRESSLER, MD, Bernard 3600 Floyd Avenue Richmond VA 23221	P	BUXTON JR., MD, Ernest Perry 5006 Cary St Richmond VA 23226	GE	CHAUDHARY, MD, Nazir Ahmad 2nd & Franklin Sts, #309 Richmond VA 23219	P	CRUIKSHANK, MD, Dwight P. Box 34, M C V Station Richmond VA 23298	OBG
BLACKMORE, MD, John Robt 5700 Old Richmond Ave Richmond VA 23226	D	BREWER, MD, William Henry Box 615, M C V Station Richmond VA 23298	DR	BUXTON, MD, Martin N. 1500 Westbrook Ave Richmond VA 23227	P	CHEN, MD, Jen-Wen 2710 Kenbury Rd Richmond VA 23225	AN	CUMMINGS, MD, Charles Edwerd 2809 North Ave Richmond VA 23222	IM
BLADES, MD, James Franklin 5806 Three Chopt Road Richmond VA 23226	GYN	BRIERE, MD, Russell Ovide Chippenharn Hospital Richmond VA 23225	CLP	BYRD, MD, Chas Wm 4906 Forest Hill Ave Richmond VA 23225	GS	CHILDREY JR., MD, Edgar Box 392 Urbenna VA 23175	OPH	CURRY, MD, Wm Lake 6823 Kensington Ave Richmond VA 23226	PD
BLAIR III, MD, Chas Jos 201 N Hamilton Street Richmond VA 23221	OPH	BROADBENT JR., MD, Reuben H. 307 St. Davids' Lane Richmond VA 23221	EM	CABILING, MD, Marino M Parham Medical Village 2101 East Parham Road Richmond VA 23228	GS	CHISHOLM, MD, Louis Randolph Box 446 Midlothian, VA 23113	FP	DABNEY JR., MD, Thos Todd 7702 Parham Road Richmond VA 23229	OPH
BLAKEY, MD, Peter P. Box 549 Midlothian, VA 23113	PD	BRODIE, MD, Owen Wingfield 1500 Westbrook Ave Richmond VA 23227	P	CADER, MD, Josephine B 2105 East Parham Rd Richmond VA 23228	PD	CHRISTIAN, MD, Chas Fletcher 2809 North Ave Richmond VA 23222	P	DABNEY, MD, Wm Taylor 3404 Sherbrook Road Richmond VA 23235	IM

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DALTON JR, MD, James B 4315 Grove Ave Richmond VA 23221	ORS	DUNN, MD, Harold Paul 2 Hillaire Lane Richmond VA 23229	PTH	FLOYD, MD, Harold Laopold 7300 Lookout Drive Richmond VA 23225	R	GIORDANO, MD, Anthony M 1403 Johnston Willis Dr Richmond, VA 23235	OTO	HADDOCK, MD, Edward Ellis 1133 W Franklin St Richmond VA 23220	GP
DANIEL III, MD, John M. 4312 Grove Avenue Richmond, VA 23221	IM	DUNN, MD, Leo James Box 34, M C V Station Richmond VA 23298	OBG	FOGEL, MD, Wm Martin 101 Gunby Drive Richmond VA 23229	DR	GIREVENDULIS, MD, Alexander K. 606 Lambeth Road Richmond, VA 23225	DR	HAGAN, MD, Christine D. P O Box 669 Goochland, VA 23063	FP
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DANIEL, MD, Jerome M. 7133 Jahnke Road Richmond, VA 23225	FP	DWYER, MD, James Henry 4906 Forest Hill Ave Richmond VA 23225	PD	FOGLE, MD, Kelly Ashworth 3500 Kensington Ave Richmond VA 23221	OPH	GLEASON, MD, Margaret Daley 6161 River Rd Apt 3 Richmond VA 23226	P	HAKALA, MD, Edwin W 2222 Monument Ave Richmond VA 23220	ORS
DANIEL, MD, Thos Moore 7129 Jahnke Rd Richmond VA 23225	TS	DYSON, MD, Maynard C. 1405 Johnston-Willis Dr Richmond VA 23235	A	FOHL, MD, Richard Bell 5855 Brems Road #503 Richmond VA 23226	D	GOLDMAN JR, MD, David Box 230, M C V Station Richmond VA 23298	IM	HAKALA, MD, Michael W 2222 Monument Ave Richmond VA 23220	ORS
DARDEN JR, MD, James Ryland 425 North Boulevard Richmond VA 23220	GS	EAGLES, MD, Wm Mc Coy 4608 Sylvan Road Richmond VA 23225	NS	FORBES, MD, Dennis Barry 4412 Park Avenua Richmond VA 23221	IM	GOLDMAN, MD, Stanley Allen 5855 Brems Rd Richmond VA 23226	CD	HAKIM, MD, Christopher A 1038 Sonnet Hill Drive Richmond VA 23236	IM
DAVID, MD, Marigall Wynne 7702 Parham Rd Richmond VA 23229	IM	EASTERLY III, MD, Harry Watkey 425 North Boulevard Richmond VA 23220	GS	FORNARIS, MD, Ernest A 10812 Whitaker Woods Rd Richmond VA 23233	IM	GOLDMANN, MD, Peter Herbert 500 Old Richmond Ave., #28 Richmond VA 23226	OPH	HALLMANN, MD, Ciamans E 1417 Johnston Willis Dr Richmond VA 23235	EM
DAVID, MD, Ronald B. 1825 Monument Avenue Richmond, VA 23220	CHN	EISENBERG, MD, Stuart J 507 Honaker Ave Richmond VA 23226	DR	FORREST, MD, David C 5855 Brems Road #207 Richmond VA 23226	OBG	GOLDSTONE, MD, Alvin I 8919 Three Chopt Road Richmond VA 23229	OTO	HALLORAN, MD, L Gregg 5855 Brems Road Richmond VA 23226	ABS
DAVIDSON, MD, Donald Dale 4315 Grove Ave Richmond VA 23221	ORS	ELLEN, MD, Joseph Harry 7151 Jahnke Road Richmond, VA 23225	OBG	FORRESTER, MD, Wm Manning T 908 N Concord Ave Richmond VA 23227	GP	GOMEZ, MD, Humberto 1500 Westbrook Avenue Richmond, VA 23227	CHP	HALLORAN, MD, Randolph M 7702 Parham Road Richmond VA 23229	IM
DAVIS JR, MD, Edward Garland 15 Buck Branch Drive Richmond VA 23233	PD	ELLIOTT, MD, Billie L Wright 2956 Hathaway Rd, #1006 Richmond, VA 23225	CHP	FOSTER JR, MD, Merritt W 414 W Franklin St Richmond VA 23220	P	GOOD, MD, John Russell 7127 Jahnke Road Richmond VA 23225	FP	HAMILTON JR, MD, Stuart H Rt 14 Box 289C Richmond VA 23231	OBG
DAVIS JR, MD, Thos Dewey 5855 Brems Rd Ste 302 Richmond VA 23226	GE	ELMORE, MD, Stanley M 7135 Jahnke Rd Richmond VA 23225	ORS	FOWLER, MD, Franklin T Box 6767 Richmond VA 23230	OS	GOODMAN, MD, Harold 3414 Walker's Ferry Rd Midlothian, VA 23113	DR	HANCOCK, MD, Wm C. 904 S Gaskins Rd Richmond VA 23233	GP
DAVIS, MD, Frederick Sterling 3500 Kensington Ave Richmond VA 23221	OPH	EPPEL III, MD, Edward M 304 Medical Arts Bldg Richmond VA 23219	FP	FRABLE, MD, Mary Ann Smith Box 115, M C V Station Richmond VA 23298	OTO	GOODMAN, MD, Peter Lewis 1400 Westwood Ave Richmond VA 23227	IM	HANZEL, MD, Jeffrey Sheldon 3603 Grove Ave Richmond VA 23221	PD
DAVIS, MD, Ronald Kenneth 417 Libbie Avenue Richmond VA 23226	GS	EVANS, MD, Eleanor Freed 14201 West Salisbury Rd Midlothian VA 23113	IM	FRABLE, MD, Wm Jackson Box 115, M C V Station Richmond VA 23219	PTH	GOODMAN, MD, Robert Paul 8200 Gaylord Road Richmond, VA 23229	IM	HARBISON, MD, John William Box 599, M C V Station Richmond VA 23298	N
DAWSON, MD, Alonzo Ray 3416 Wythe Ave Richmond VA 23221	PM	EVANS, MD, Martin Terry 7702 Parham Road Richmond, VA 23229	GS	FRANCO JR, MD, Andres P 701 West Grace Street Richmond VA 23220	PTH	GOODNER, MD, John Wood Medical Science Bldg, #18-D Richmond VA 23226	OBG	HARD JR, MD, Richard C. Box 662, M C V Station Richmond VA 23298	P
DECKER, MD, Henry Chesley 1400 Westwood Ave Ste 110 Richmond VA 23227	IM	EWART, MD, Geo E 8720 Chippaham Rd Richmond VA 23235	PUD	FRATKIN, MD, Malvin Joel Box 481, M C V Station Richmond VA 23298	R	GOPLERUD, MD, Dean Roy Box 34, M C V Station Richmond VA 23298	GYN	HARKRADER, MD, James Collin 121 Wyck Street Richmond VA 23225	OPH
DECKER, MD, Michael John 2004 Brems Road Richmond VA 23226	US	FAIRLY JR, MD, John L 8256 Halstead Rd Richmond VA 23235	PUD	FREDERICK, MD, Louis Arnold 209 Clovelly Rd Richmond VA 23221	U	GORMAN, MD, Jerome Davis 3900 Chamberlayne Ave Richmond VA 23227	GP	HARLER SR, MD, John Ashby 4312 Grove Avenue Richmond VA 23221	IM
DEEP, MD, Anthony Abraham 2002 Brems Rd Ste 201 Richmond VA 23226	OBG	FALLON, MD, Harold Jos Box 663, M C V Station Richmond VA 23298	GE	FREUND, MD, Jack 700 W Grace St, #101 Richmond VA 23220	IM	GOSPODNETIC, MD, Marjian 7153 Jahnke Road Richmond VA 23225	OBG	HARLFINGER, MD, Erwin Harbert 3715 Chellowe Rd Richmond VA 23225	OBG
DEEP, MD, Wm Danl 6912 Three Chopt Rd Richmond VA 23226	IM	FALLS JR, MD, William Franklin 2417 Scarsborough Drive Richmond, VA 23235	IM	FREY, MD, Allan Arthur 9319 Westmoor Circle Richmond VA 23229	DR	GOTTWALD, MD, William 7239 Cherokee Road Richmond, VA 23225	D	HARP, MD, William Lee 8109 Kingston Road Richmond VA 23229	P
DEL SORDO, MD, Andrew A 2500 Pocoshock Pl Richmond VA 23235	OS	FANTL, MD, Juan Andres E Box 34, M C V Station Richmond VA 23298	OBG	FRIEDENBERG, MD, Milton David 5855 Brems Road Ste 303 Richmond VA 23226	P	GRADY, MD, Ann Elizabeth 325 Oak Lane Richmond, VA 23226	IM	HARRELLSON, MD, Austin B 1805 Monument Ave Richmond VA 23220	N
DEMAURIZI, MD, Lorenzo A Route 1, Box 465-A Ashland, VA 23005	GP	FARLEY JR, MD, Emerson Dale 2002 Brems Rd Lower Level Richmond VA 23226	RHU	FRIERSON JR, MD, John Hugh 4701 S Springs Ct Midlothian, VA 23113	R	GRAHAM JR, MD, Ota Trevilla 3415 Floyd Ave Richmond VA 23221	FP	HARRINGTON, MD, William G 4906 Forast Hill Ave, #114 Richmond, VA 23225	FP
DENT, MD, Roy Wm 45 Old Mill Rd Richmond VA 23226	OS	FARNSWORTH, MD, David I 1711 Bellevue Ave, #D 114 Richmond VA 23227	IM	FRISCHKORN JR, MD, Hunter B 18 Bridgeway Rd Richmond VA 23226	R	GRAHAM, MD, A Stephens 811 Arlington Circle Richmond VA 23229	GS	HARRIS JR, MD, Campbell 1805 Monument Ave, #506 Richmond VA 23220	AN
DETHMERS, MD, Daniel A. 2911 Grove Avenue Richmond, VA 23221	ORS	FAUNCE, DO, Howard F 102 N. Moreland Rd Richmond VA 23229	OST	GALESKI III, MD, Joseph 7702 Parham Road Richmond, VA 23229	IM	GREENBERG, MD, David Jeremiah 1805 Monument Ave Richmond VA 23220	IM	HARRIS JR, MD, Wm Henry 108 Windsor Way Richmond VA 23221	IM
DEYERLE, MD, William Minor 2222 Monument Ave Richmond VA 23220	ORS	FEORE, MD, John Colman 901 Hoaks Road Richmond VA 23225	OBG	GALSTON, MD, Herbert Harold 2038 Monument Ave Richmond VA 23220	R	GREENFIELD, MD, Lazar John Box 645, M C V Station Richmond VA 23298	TS	HARRISON, MD, Henry Tucker 5801 Brems Rd Emergency Dept Richmond VA 23226	EM
DIEHL JR, MD, Earl Henry 900 N Hamilton Street Richmond VA 23221	GE	FERRAR, MD, Wm Lewis Parham Medical Center Richmond VA 23229	IM	GARDNER, MD, Shockley D Rt 1 Box 123 Richmond VA 23231	GP	GREGORY, MD, James Stewart 2000 Brems Rd #201B Richmond VA 23226	CRS	HARRISON, MD, Jacquelin M 7129 Jahnke Rd Richmond VA 23225	GS
DINEEN, MD, Mary Kay Box 34, M C V Station Richmond, VA 23298	OBG	FIDLER, MD, Robt Young 7601 Forest Ave, #221 Richmond VA 23229	PD	GAYLE, MD, Sigsby Warren 7115 Jahnke Rd Richmond VA 23225	IM	GRIFFITH, MD, Kenneth Cornell 7601 Forest Avenue Richmond VA 23229	CD	HARTLEY, MD, A. Howland 5801 Brems Rd, #507 Richmond, VA 23226	PD
DINGLEDINE, MD, Wm S 5500 Monument Ave, #1 Richmond VA 23226	IM	FIEDLER, MD, Adam J 901 Hoaks Road Richmond, VA 23225	OBG	GAZALA, MD, Jos Richard 4508 Grove Ave Richmond VA 23221	OPH	GRINMAN, MD, Richardson Box 27401 Richmond VA 23279	PUD	HARWOOD, MD, Chas Pinchback 6 E Williamsburg Rd Sandston VA 23150	GP
DIXON, MD, Leon M 1951 Reymet Rd Richmond VA 23234	IM	FIERRO, MD, Anthony Parham Medical Center Richmond, VA 23229	FP	GELRUD, MD, Louis Gerald 5855 Brems Road, #208 Richmond VA 23226	IM	GRINMAN, MD, Wm C 7110 Club Vista Lane Richmond VA 23229	IM	HASTILLO, MD, Andrea Karen 14031 Elmstead Road Midlothian, VA 23113	CD
DODD, MD, Richard Wine 204 N Hamilton Richmond VA 23221	OTO	FIERRO, MD, Marcella F 9 North 14th Street Richmond VA 23219	FOP	GENTILE, MD, Francis G 13600 Trilithon Rd Midlothian, VA 23113	OBG	GRUNER, MD, George 13421 Torrington Drive Midlothian, VA 23113	NS	HAYDEN, MD, Geo Douglas 331 Oak Lane Richmond VA 23226	OTO
DODSON JR, MD, Austin Ingram 2002 Brems Rd Ste 202 Richmond VA 23226	U	FIERRO, MD, Robert J 2006 Brems Road, #202 Richmond VA 23226	OBG	GERSTEN, MD, Enrique Box 662, M C V Station Richmond VA 23298	PTH	GUERRY III, MD, Du Pont 2015 Monument Ave Richmond VA 23220	OPH	HAYNES JR, MD, Boyd W Box 661, M C V Station Richmond VA 23298	GS
DOOLEY, MD, Mark C. 900 N. Hamilton Street Richmond, VA 23221	IM	FISHER JR, MD, Hugh P. 1536 Clarkson Road, #B Richmond VA 23224	IM	GHAPHERY, MD, James Louis 1443 Johnston Willis Dr Richmond VA 23235	AN	GUERRY, MD, Richard Kennon 2015 Monument Ave Richmond VA 23220	OPH	HAZRA, MD, Tapan A Box 533, M C V Station Richmond VA 23298	TR
DOS SANTOS III, MD, Joao G 2 Glenbrooke Circle West Richmond VA 23229	CLP	FISHER, MD, Dorothy Gwendoline 9202 Waterloo Ct Richmond VA 23229	P	GILL, MD, Fleming W 2924 Chamberlayne Ave Richmond VA 23222	GP	GURIGUIS, MD, Habib Habib 9925 Aldermead Ct Bon Air VA 23235	EM	HENCEROOTH II, MD, William D 2222 Monument Ave Richmond VA 23220	ORS
DRAKE, MD, John Edward 2002 Brems Lower Level Richmond, VA 23226	CD	FISHER, MD, Lyman Mc Arthur 9202 Waterloo Court Richmond VA 23229	CLP	GILL, MD, James T 108 Mannheim Drive Ashland, VA 23005	GP	GUTHRIE, MD, Michael J 5311 Patterson Ave Richmond, VA 23226	P	HENLEY JR, MD, Robert W P O Box 13470, #D Richmond VA 23225	IM
DRUMMOND JR, MD, Chas Stitt 7702 Parham Rd Richmond VA 23229	GS	FITCH, MD, Willard M. 13520 Heathbrook Road Midlothian VA 23113	R	GILL, MD, John A 7123 Jahnke Rd Richmond VA 23225	AN	GUTER, MD, Marvin P O Box 29246 Richmond VA 23229	R	HENNESSEY IV, MD, John J. 3249 Summitbrook Dr Richmond, VA 23235	N
DUCK, MD, George Bryan 2002 Brems Road, #202 Richmond, VA 23226	U	FITZGERALD, MD, John E. 7601 Forest Ave, #334 Richmond, VA 23229	CD	GILLESPIE, MD, Harold E 1827 Grove Ave Richmond VA 23220	GPM	GWATHMEY, MD, Owen 1400 Westwood Ave, #101 Richmond VA 23227	CD	HERMAN, MD, Bernard D 2821 Parham Road Richmond VA 23229	PO
DULEY, MD, Robt Kingston 2913 Park Avenue Richmond VA 23221	GP	FITZTHUGH, MD, William Garth 2016 Monument Ave Richmond VA 23220	OBG	GILLESPIE, MD, Lester Langdon 8 Rock Garden Lane Richmond, VA 23228	AN	HACKLER, MD, Robt Hardin 12105 Waxwood Place Richmond VA 23235	U	HERMANN, MD, Ernest Conrad 3424 Pump Rd Richmond VA 23233	GP

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HEYNER, MD, Gregory James 8133 Forest Hill Avenue Richmond VA 23235	OPH	ISAACS, MD, Edward Richard 10806 Chipewyan Dr Richmond VA 23233	N	JORDAN, MD, Wm R 16314 Amberwood Rd Dallas, TX 75248	IM	KITCES, MD, Eileen C. 1810 Monument Ave Richmond VA 23220	D	LINDEMANN, MD, Lillian C 2223 Monument Ave Richmond VA 23220	CHP
HICKMAN, MD, Clifton C. 1423 Johnston Willis Dr Richmond VA 23235	OTO	JACEY, MD, Robt Wayne 2821 Parham Road Richmond VA 23229	OPH	KAHN, MD, Howard Donald 1400 Westwood Avenue Richmond, VA 23227	IM	KLEIN, MD, Arthur 2016 Monument Ave Richmond VA 23220	R	LIPPER, MD, Maurice H Box 615, M C V Station Richmond VA 23298	2016
HIGGINS JR., MD, Wm Harrison 3540 Floyd Ave Richmond VA 23221	IM	JACKSON JR., MD, Gustavus V 5855 Brems Road Richmond VA 23226	OM	KALLAR, MD, Surinder Kaur Box 695, M C V Station Richmond VA 23298	AN	KLEIN, MD, Frederick A. Box 118, M C V Station Richmond VA 23298	U	LITCHFIELD, MD, David Lee 7702 Parham Rd Richmond VA 23229	NM
HILL, MD, John Edward 2002 Brems Rd Ste 202 Richmond VA 23226	U	JACKSON, MD, Hunter S 213 Banbury Rd Richmond VA 23221	PS	KANNAN, MD, Mickael M. 5901 Lakeside Ave Richmond, VA 23228	ID	KNAYS, MD, George A. 8921 Three Chopt Rd Richmond VA 23229	GS	LOGANATHAN, MD, Sri Tharan 8301 Trubus Rd Richmond VA 23235	PD
HILL, MD, Wm R 609 W Grace St Richmond VA 23220	GS	JACKSON, MD, Richard A 317 E Clay Street Richmond, VA 23240	IM	KAPIL, MD, Belvir L 1400 Westwood Ave Richmond VA 23227	GS	KNIGHT, MD, Frank Sutton 6823 Kensington Ave Richmond VA 23226	OBG	LONGACHER JR., MD, Jos Wm 7702 Parham Road Richmond VA 23229	IM
HLADYS, MD, Jacob J 2111 W Main St Richmond VA 23220	P	JAFFE, MD, Michael 9606 Patterson Ave Richmond VA 23229	PD	KATCHINOFF, MD, Barry L. 7131 Jahne Road Richmond, VA 23225	N	KNIGHT, MD, Yvonne 2809 North Ave Richmond, VA 23222	D	LONGAN JR., MD, Robt C 14 Westham Green Richmond VA 23229	P
HOFF, MD, Ebbe Curtis 117 Gaymont Road Richmond VA 23229	P	JAFFE, MD, Stephen L 2105 East Parham Road Richmond VA 23228	N	KAY, MD, Saul 322 Charming Rd Richmond VA 23226	PTH	KOLLER, MD, Stephen Rice 5855 Brems Rd Richmond, VA 23226	RHU	LORDI, MD, William M. 8635 Mayland Drive Richmond VA 23229	CHP
HOFFMAN, MD, Ivan Bruce 3600 W Broad St, #444 Richmond VA 23230	P	JAMES III, MD, G. Watson 2401 Burroughs Ct Richmond VA 23235	HEM	KAZI, MD, Seyed 5001 W Village Gr, #109 Midlothian, VA 23113	PTH	KONERDING, MD, Hazel S. 5855 Brems Road, Suite 503 Richmond VA 23226	D	LOVINGER, MD, Robert D. 6716 Patterson Ave Richmond VA 23226	EN
HOFFMAN, MD, Robt Arnold 11803 North Downs Sq Richmond VA 23233	PD	JAMES IV, MD, George W 4101 Johnston Willis Dr Richmond, VA 23235	IM	KEBLUSEK, MD, Charles W. 2803 Mc Rae Road Richmond, VA 23235	OBG	KONERDING, MD, Karsten F 7702 Parham Road Richmond VA 23229	DR	LUBLIN, MD, Bernard A 8600 Quicocasin Rd, #103 Richmond VA 23229	ORS
HOGGE, MD, Randolph H Longview Manakin VA 23103	GYN	JAMES, MD, Charles M 7601 Forest Ave Richmond VA 23229	ORS	KEENAN, MD, Richard Leo Box 695 M C V Station Richmond VA 23298	AN	KONTOS, MD, Hermes Apostolou Box 281, M C V Station Richmond VA 23298	CD	LYNCH, MD, John P 7702 Parham Road Richmond VA 23229	GER
HOKKE, MD, Harry Franklin 2621 Grove Ave Richmond VA 23220	PTH	JANES JR., MD, John R 5855 Brems Road Richmond, VA 23226	AN	KEENAN, MD, Robert Edward 1211 Sherwood Avenue Richmond, VA 23220	FP	KOONTZ, MD, Warren Woodson Box 118, M C V Station Richmond VA 23298	U	MAC MILLAN, MD, David W. 2821 Parham Road, #102 Richmond VA 23229	OPH
HOLLAND, MD, Henry Davis 3900 Monument Ave, #A Richmond VA 23230	P	JARRELL, MD, Shelby Edward 1400 Westwood Ave Richmond VA 23227	OBG	KELL JR., MD, Joseph F 7702 Parham Road Richmond VA 23229	NS	KOWLER, MD, Daniel Edward 5855 Brems Rd, Ste 404 Richmond VA 23226	P	MAC MILLAN, MD, James M 306 Gunby Drive Richmond VA 23229	OM
HOLLAND, MD, William Elisha 1602 Skipwith Rd Richmond VA 23229	CD	JARRETT, MD, John Tellman 301 Desota Dr Richmond VA 23229	GS	KELLET II, MD, Gordon N 8710 Chocataw Rd Richmond VA 23235	PD	KOZIOL, MD, Isaac 5855 Brems Rd, #204 Richmond, VA 23226	U	MACK, MD, Theodore R 8703 Standish Lane Richmond VA 23229	OBG
HORSLEY, MD, John S Box 11, M C V Station Richmond VA 23298	GS	JESSEE JR., MD, Edgar Forrest 1411 Johnston Willis Dr Richmond VA 23235	RHU	KELLEY JR., MD, William 8921 Three Chopt Road Richmond, VA 23229	GS	KRAMER, MD, William 5801 Brems Rd., Leb Richmond, VA 23226	PTH	MACMILLAN, MD, Ralph Victor P O Box 7314 Richmond, VA 23221	GP
HOUSER JR., MD, Aubrey A 105 Kennondale Lane Richmond VA 23226	DR	JESSEE, MD, Robt W Route 2, Box 12-C Hanover, VA 23069	GPM	KELLY III, MD, John Jackson 8510 Academy Road Richmond VA 23229	IM	KRAUS, MD, Shana James P O Box 608 Providence Forge, VA 23141	GP	MACYS, MD, Joseph R 2911 Grova Ave Richmond VA 23221	ORS
HOWELL, MD, Hugh Richard 1415 Johnston Willis Dr Richmond VA 23235	N	JESSEE, MD, Samuel D. 5001 W Village Green Dr. Midlothian, VA 23113	ORS	KELLY JR., MD, Frank R 2031 Monument Ave Richmond VA 23220	IM	KREBS, MD, Hans-Bartold Box 34, M C V Station Richmond, VA 23298	OBG	MADGE, MD, Gordon Evans Box 473, M C V Station Richmond VA 23298	FOP
HOWREN JR., MD, Harry H 8710 Chocataw Rd Richmond VA 23235	GYN	JESSUP, MD, Douglas E. 4315 Grove Avenue Richmond, VA 23221	ORS	KELLY JR., MD, Leonard Wm 7103 A Jahne Road Richmond VA 23225	D	KREISLER, MD, Leslie S 5855 Brems Rd Ste 203 Richmond VA 23226	OTO	MAIZELS, MD, Max Sam 2016 Monument Ave Richmond VA 23220	OBG
HRANOWSKY, MD, Nicholas P 4100 Taylor Dr Richmond VA 23235	AN	JETER, MD, William Richard Parham & Quicocasin Roads Richmond, VA 23226	FP	KEMP JR., MD, Verbon Eric 7601 Forest Avenue Suite 334 Richmond, VA 23229	CD	KRETTEK, MD, John E. Box 631, M C V Station Richmond, VA 23298	NS	MAKAROWSKY, MD, Eugene 4106 Forest Circle Richmond VA 23225	P
HUBERT, MD, Juergen 26 Lower Tuckahoe Rd W Richmond VA 23233	AN	JOHNS, MD, Thos Nelson Page 6305 Towena Rd Richmond VA 23226	GS	KENDIG JR., MD, Edwin L 5855 Bramo Road, #507 Richmond VA 23226	PD	KRICKOVIC, MD, Milan Piersol 2000 Brems Road, #101 Richmond VA 23226	CD	MALTESE, MD, Francas Anna 9409 Avalon Dr Richmond VA 23229	OBG
HUDGENS, MD, Robt Oscar 7159 Jahne Road Richmond VA 23225	PD	JOHNS, MD, Wm A 1401 Johnston-Willis Dr Richmond VA 23235	GS	KENDRICK JR., MD, John Fox 7601 Forest Ave, #336 Richmond VA 23229	NS	KROLL, MD, Ronald Neil 2505 Pocoshock Place Richmond, VA 23235	NEP	MANDEL, MD, Michael David 5855 Brems Ave Ste 403 Richmond VA 23226	IM
HUDGINS, MD, Earl Maxwell 411 Kilmarnock Dr Richmond VA 23229	D	JOHNSON, MD, Bruce E 2000 Bramo Rd, #105 Richmond VA 23226	OBG	KENLEY, MD, James B 3010 Kenbury Rd Richmond VA 23235	PH	KRONFOL, MD, Nouhad O. Box 160, M C V Station Richmond, VA 23298	IM	MANETZ, MD, Chleras E 2721 Arrandall Road Midlothian VA 23113	R
HUDSON, MD, Gwendolyn S 1905 Woodbine Road Richmond VA 23225	OBG	JOHNSON, MD, Geo Wm 7109 Jahne Road Richmond, VA 23225	NS	KERN, MD, Marguerite Ann Route 3, Box 556 Ashland, VA 23005	PD	KUPERMINE JR., MD, Mario 5855 Brems Rd Ste 506 Richmond VA 23226	ON	MANGUS, MD, Julien Edward 1602 Treboay Avenue Richmond VA 23226	FP
HUEROGO, MD, Eduardo R. 10111 Deepwood Circle Richmond, VA 23233	CHP	JOHNSON, MD, Hal S 202 Wood Rd Richmond VA 23229	OPH	KERNODLE, MD, Judith M. 1101 W 43rd Street Richmond VA 23225	P	KUPERMINE, MD, Denis S. 301 Roslyn Rd Richmond VA 23226	PD	MANN, MD, H. Thompson 1617 Monument Ave, #202 Richmond, VA 23220	GP
HUGHES, MD, David G 1441 Johnston Willis Dr Richmond, VA 23235	CD	JOHNSTON JR., MD, Chas L Box 597, M C V Station Richmond VA 23298	CLP	KERNODLE, MD, Wm Dwight 9925 Kingussle Lane Richmond VA 23236	P	KURUP, MD, Nankoth G 1400 Westwood Ave, #101 Richmond VA 23227	GS	MANSON, MD, R Campbell 1832 Monument Ave Richmond VA 23220	D
HULCHER, MD, Julius C. 11804 North Downs Square Richmond VA 23233	OTO	JOHNSTON, MD, Wm Burton 3602 Monument Ave Richmond VA 23230	PD	KEY JR., MD, Wendell Wayne 6610 W. Broad Street Richmond VA 23230	IM	LA FRATTA, MD, Carl W 8664 Trubus Rd Richmond VA 23235	PM	MARKHAM, MD, J. David 5855 Brems Road Richmond VA 23226	IM
HULLEY JR., MD, Levi W 8908 River Road Richmond VA 23229	OS	JOHNSTONE, MD, William T 8919 Three Chopt Road Richmond VA 23229	ORS	KILUK, MD, Kenneth Ignatius 7109 Jahne Road Richmond, VA 23225	NS	LANKFORD, MD, Harvey V. 2002 Brems Road Richmond, VA 23226	EN	MARKOWITZ JR., MD, Martin 808 N Hamilton St Richmond VA 23221	GS
HUNT, MD, Patricia A 2956 Hathaway Rd, Apt 1111 Richmond, VA 23225	GPM	JONES JR., MD, Beverly 13902 Commodore Pt Rd Midlothian, VA 23113	AN	KIM JR., MD, Jin Tek 11400 Poplar Ridge Rd Richmond VA 23236	P	LAWRENCE JR., MD, Walter Box 11, M C V Station Richmond VA 23298	ND	MARKOWITZ, MD, Micheal Paul 1908 Windingridge Dr Richmond VA 23233	IM
HUNTER, MD, Samuel B 1500 North 28th Street Richmond, VA 23223	PTH	JONES JR., MD, Wm Russell 412 Libbie Ave Richmond VA 23226	U	KING JR., MD, Robert Gerlend 2801 Parham Road, #104 Richmond VA 23229	OPH	LE FON, MD, James C 2101 Maplewood Rd Richmond VA 23228	GS	MARSLAND, MD, David Wilson 11800 Bollingbrook Drive Richmond VA 23235	PD
HURT, MD, Waverly Glenn Box 34, M C V Station Richmond VA 23298	OBG	JONES, MD, Basil B 3103 Sunset Ave Richmond VA 23221	PD	KING, MD, Donald Perry 7702 Parham Road Richmond VA 23229	R	LEE, MD, Hyung Mo Box 57, M C V Station Richmond VA 23298	GS	MARTIN JR., MD, Berkaley H 1805 Monument Ave Richmond VA 23220	OPH
HUTCHER, MD, Neil Edward 4908 Monument Ave, #102 Richmond VA 23230	GS	JONES, MD, Donald Kenneth Box 1797 Richmond VA 23214	P	KING, MD, Elmer Richard 641 Farnham Drive Richmond VA 23235	TR	LEE, MD, Lucia Soondong 1200 Loch Lomond Court Richmond VA 23221	AN	MARTIN JR., MD, George W. 10910 Pegwell Drive Midlothian, VA 23113	DR
HUTTON, MD, Chas Frederick 2237 Brookwood Rd Richmond VA 23235	DR	JONES, MD, Geo Robt 6 Glenbrooke Circle E Richmond VA 23229	OBG	KING, MD, Joseph Willett 3001 Fifth Avenue Richmond, VA 23222	CHP	LEHMANN III, MD, Ilse 5059 Warwick Rd Richmond VA 23224	GP	MARTIN, MD, Wm Watkins 2201 Grove Ave Richmond VA 23220	IM
HYSLUP, MD, John Wesley 3536 Grove Avenue Richmond, VA 23221	GS	JONES, MD, John P Rt 1, Box 3 Aa Hanover, VA 23069	PD	KIPREOS, MD, Theophilos H 4801 Bromley Lane Richmond VA 23226	*	LENETT, MD, Stephen David 2327 Thousand Oaks Drive Richmond VA 23229	EM	MARTIROSIAN, MD, Edward D 7601 Forest Ave #340 Richmond VA 23229	CD
IATRIDIS, MD, Angelos T B Conrol 109 Governor St Richmond VA 23219	PUD	JONES, MD, John R 18121 Wakehurst Dr Richmond VA 23236	AN	KIRCHMIE, MD, Raymond S 7000 Patterson Ave Richmond VA 23226	PD	LEVERTY, MD, Alexander P 5204 Patterson Ave Richmond VA 23226	PD	MASICA, MD, Daniel N Route 6, Box 61-X Powhatan, VA 23139	FP
IRBY, MD, Edward Claiborne 225 Ross Road Richmond VA 23229	OM	JONES, MD, Steven H. 4315 Grove Ave Richmond, VA 23221	ORS	KIRKLAND, MD, Richard Horace 8002 University Dr Richmond VA 23229	END	LEVINE, MD, Jay Michael Box 26783 Richmond, VA 23261	R	MASSEY, MD, Chas Webster 7Buckeye? Rt #2, Box 99 Mechanicville, VA 23111	R
IRBY, MD, W Robt Box 647, M C V Station Richmond VA 23298	RHU	JONES, MD, Wm Collins 2809 North Ave Richmond VA 23222	OBG	KIRKPATRICK, MD, Barry V Box 276, M C V Station Richmond VA 23298	PD	LEVINSON, MD, Harold Jay 7107 Jahne Road Richmond VA 23225	CDS	MASSIE, MD, F Stanford 5855 Brems Rd Rm 409 Richmond VA 23226	AI
IRWIN, MD, Charles Fayette 7702 Parham Road Richmond VA 23229	FP	JORDAN JR., MD, Wm Pritchard Box 296, M C V Station Richmond VA 23298	D	KISHORE, MD, P.R.S. Box 615, M C V Station Richmond VA 23298	DR	LEWIS, MD, Richard Gordon P O Box 13470, #D Richmond, VA 23225	CD	MATHEWS, MD, Emmett C 204 N Hamilton St Ste 5 Richmond VA 23221	IM

\* = AMA membership



<b>MATTHEWS</b> , MD, Richard Eugene 5 West Nine Mile Road Highland Springs VA 23075	EM	<b>MELZIG</b> , MD, Eric Perry 8921 Three Chopt Rd Richmond VA 23229	ABS	<b>MULLINS</b> , MD, Maurice Francis 211 Massie Road Richmond VA 23221	R	<b>PACKER</b> , MD, Bernard D 5311 Patterson Ave Richmond VA 23226	ORS	<b>PRITCHARD</b> , MD, Geo Emmett 5001 Grove Ave Richmond VA 23226	CD
<b>MAUCK JR</b> , MD, Henry P 113 Oxford Circle West Richmond VA 23221	PDC	<b>MENDEZ</b> , MD, Manuel Diaz 5500 Monument Ave Suite O Richmond VA 23226	GS	<b>MUREN JR</b> , MD, Orhan Box 50, M C V Station Richmond VA 23298	PUD	<b>PAGE</b> , MD, Maysville J Owens 2904 Rugby Rd Richmond VA 23221	PD	<b>PROCTOR</b> , MD, Jack Douglas Box 284, M C V Station Richmond VA 23298	IM
<b>MAUCK</b> , MD, William R 7601 Forest Ave Richmond VA 23229	ORS	<b>MERCER</b> , MD, H. Richard P O Box 11182 Richmond VA 23230	FP	<b>MURRELL JR</b> , MD, Thos W 17 E Grace St Richmond VA 23219	D	<b>PAGE</b> , MD, Sidney G 2904 Rugby Rd Richmond VA 23221	PM	<b>PROCTOR</b> , MD, William Franklin 7702 Parham Rd Mc Guire Clinic Richmond VA 23229	DR
<b>MAY III</b> , MD, James Terrell 5855 Brems Rd, #506 Richmond VA 23226	ON	<b>MERCHANT III</b> , MD, Wilson C. 8809 Wishart Road Richmond VA 23229	U	<b>NACHMAN</b> , MD, Herman M 808 N Hamilton St Richmond VA 23221	ORS	<b>PAK</b> , MD, Yong Kun 2903 Scherer Dr Richmond VA 23235	AN	<b>PROUD</b> , MD, Virginia Kent 2810 West Brigstock Road Midlothian, VA 23113	PD
<b>MAY JR</b> , MD, Virgil R 2222 Monument Ave Richmond VA 23220	ORS	<b>MERRILL</b> , MD, Cynthia Westneat 4908 Monument Avenue Richmond VA 23220	FP	<b>NAKONECZNA</b> , MD, Irene Box 662, M C V Station Richmond VA 23298	PTH	<b>PANCOAST</b> , MD, James White 417 Libbie Avenue Richmond VA 23226	US	<b>PUSTER JR</b> , MD, Geo Valentine 3300 West Grove Avenue Chester, VA 23831	US
<b>MAYER</b> , MD, Walter 2004 Brems Rd Ste 100 Richmond VA 23226	OPH	<b>MEYERHOFF</b> , MD, Geo Edward 1617 Monument Avenue Richmond VA 23220	IM	<b>NALLS III</b> , MD, Cecil A 414 West Franklin St Richmond VA 23220	P	<b>PARK</b> , MD, Herbert Wm 7702 Parham Road Richmond VA 23229	PM	<b>RABHAN</b> , MD, Nathan H 8921-23 Three Chopt Rd, #101 Richmond VA 23229	OBG
<b>MAYO</b> , MD, Fitzhugh Box 251 Richmond VA 23298	FP	<b>MEYERS</b> , MD, John Fredrick 8919 Three Chopt Road Richmond VA 23229	ORS	<b>NASSIRI</b> , MD, Saeed K 213 Chickahominy Bluffs Rd Richmond VA 23227	IM	<b>PARK</b> , MD, Sung Chull Stuart Circle Hospital Richmond VA 23220	PTH	<b>RABHAN</b> , MD, Walter N 8919 Three Chopt Rd Richmond VA 23229	ORS
<b>MC ALLISTER SR</b> , MD, Russell 1016 W Franklin Richmond VA 23220	FP	<b>MICHAUX</b> , MD, Richard A 425 North Boulevard Richmond VA 23220	GYN	<b>NATVIG</b> , MD, Ralph Andreas 3536 Grove Ave Richmond VA 23221	GS	<b>PARKER</b> , MD, Jos C Box 14557 Richmond VA 23221	GYN	<b>RADOW</b> , MD, Scott K. 2311 Persimmon Trek Richmond VA 23233	PUD
<b>MC CAHILL</b> , MD, Thos D 6108 Lee Ave Mechanicsville VA 23111	GP	<b>MIDDLETON JR</b> , MD, Paul 1423 Johnston Willis Dr Richmond VA 23235	OTO	<b>NEAL JR</b> , MD, M Pinson 4607 Stratford Rd Richmond VA 23225	DR	<b>PARTRIDGE</b> , MD, John Robt 7151 Jahnke Road Richmond VA 23225	OBG	<b>RAFII</b> , MD, Amir 3 Raven Rock Court Richmond VA 23229	AN
<b>MC CUE JR</b> , MD, Howard M Life Ins Co Va Box 27601 Richmond VA 23261	OM	<b>MIDIS</b> , MD, Panos M 9903 Kingsbridge Rd Richmond VA 23233	AN	<b>NELSON</b> , MD, Bobby Wayne 500 North Boulevard Richmond VA 23220	P	<b>PASTORE</b> , MD, Peter A N 5503 Riverside Dr Richmond VA 23225	OTO	<b>RAGLAND JR</b> , MD, Stuart 2604 Kensington Ave Richmond VA 23220	CD
<b>MC CUE</b> , MD, Carolyn Moore Box 272, M C V Station Richmond VA 23298	CD	<b>MILLER JR</b> , MD, Grayson Brownlee 3700 Whitewood Road Richmond VA 23235	IM	<b>NELSON</b> , MD, C M Kinloch 5224 Monument Ave Richmond VA 23226	U	<b>PATEL</b> , MD, Nagindas L 8116 Surreywood Dr Richmond VA 23235	P	<b>RAH</b> , MD, Kang H 1809 Winderidge Dr Richmond VA 23233	AN
<b>MC CUTCHEON JR</b> , MD, Randolph 7140 Hull St Rd Richmond VA 23235	FP	<b>MILLER</b> , MD, James Wesley 1417 Johnston Willis Dr Richmond VA 23235	FP	<b>NELSON</b> , MD, Kinloch 4205 Dover Road Richmond VA 23221	IM	<b>PEARCE</b> , MD, Leroy S 3530 Falstone Road Richmond VA 23234	PTH	<b>RAHAL</b> , MD, Frederick 5901 Lakeside Ave Richmond VA 23228	PD
<b>MC DANIEL</b> , MD, Leroy S 8709 Forest Hill Ave Bon Air VA 23235	FP	<b>MILLER</b> , MD, Michael J 7702 Parham Road Richmond VA 23229	RHU	<b>NEMUTH</b> , MD, Harold Isaac 2012 Monument Ave Richmond VA 23220	GP	<b>PEARS</b> , MD, Jenifer D 3006 Seminary Ave Richmond VA 23227	PD	<b>RAMSEY JR</b> , MD, Edward J. 900 N. Hamilton Richmond VA 23221	GE
<b>MC DONOUGH</b> , MD, Wm Wallace 209 Culpeper Road Richmond VA 23229	PYA	<b>MILLS</b> , MD, Julia Hines Box 549 Midlothian, VA 23113	PD	<b>NEWMAN</b> , MD, Eugene Michael 5855 Brems Rd Ste 201 Richmond VA 23226	END	<b>PELLOCK</b> , MD, John Michael Box 211, M C V Station Richmond VA 23298	CHN	<b>RANDOLPH</b> , MD, Bruce L 4416 Monument Ave Richmond VA 23230	OBG
<b>MC DOWELL</b> , MD, Charles L 2911 Grove Ave Richmond VA 23221	HS	<b>MINOR</b> , MD, Philip Lee Allen 3536 Grove Ave Richmond VA 23221	OBG	<b>NEWSOME JR</b> , MD, Heber H Box 465, M C V Station Richmond VA 23298	GS	<b>PEOTROWSKI</b> , MD, Richard Francis 3502 Walkers Ferry Road Midlothian, VA 23113	AN	<b>RAO</b> , MD, Jaikar Sudhakar 8200 Capelwood Drive Richmond VA 23235	EM
<b>MC ENTEE</b> , MD, James Phillip 1606 Monmouth Pl W Richmond VA 23233	OBG	<b>MITCHELL JR</b> , MD, Robt E 7601 Forest Ave St 332 Richmond VA 23229	IM	<b>NIATZ</b> , MD, Salullah Khan 1900 Windingridge Dr Richmond VA 23233	P	<b>PEPLE JR</b> , MD, Wm Lowndes 26 Rio Vista Lane Richmond VA 23226	GS	<b>RAWLS</b> , MD, John Ashburn 3536 Grove Ave Richmond VA 23221	GS
<b>MC ENTEE</b> , MD, Robt Bernard Parham Med Ctr Richmond VA 23229	IM	<b>MOFFATT</b> , MD, Thomas Lee 6740 Forest Hill Ave, #102 Richmond VA 23225	IM	<b>NOFSINGER</b> , MD, Dennis E 13303 Midlothian Tmpk, #A Midlothian, VA 23113	PD	<b>PEPPLE</b> , MD, Allen W 311 Medical Arts Bldg Richmond VA 23219	D	<b>RAY</b> , MD, Edward Scott 7604 Hampshire Road Richmond VA 23229	PUD
<b>MC ENTIRE</b> , MD, Wesley Edward 8923 Three Chopt Rd, #302 Richmond VA 23229	P	<b>MOHAGHEGHI</b> , MD, Hassan P O Box 5426 Richmond VA 23220	A	<b>NOGI</b> , MD, Jay 2924 Brook Road Richmond VA 23220	ORS	<b>PEREZ</b> , MD, Justo T. P O Box 267 Midlothian VA 23113	IM	<b>REAMS</b> , MD, B. Thomas 103 Cyril Lane Richmond VA 23229	D
<b>MC GEHEE JR</b> , MD, Read F 5855 Brems Rd, #509 Richmond VA 23226	PUD	<b>MOLLEN</b> , MD, Edward Leigh 5855 Brems Rd, #409 Richmond VA 23226	PDA	<b>NOTTINGHAM JR</b> , MD, Maurice 7115 Jahnke Road Richmond VA 23225	IM	<b>PERKINS</b> , MD, Edward W 300 W Franklin St Richmond VA 23220	US	<b>REBAN III</b> , MD, John 7730 Kenmore Circle Richmond VA 23225	AN
<b>MC GHEE</b> , MD, Judith F. 3109 Fox Chase Drive Midlothian, VA 23113	PD	<b>MONCURE</b> , MD, Wm B 608 Lee Medical Bldg Richmond VA 23220	AN	<b>NOVO</b> , MD, Antonio Miguel 5801 Pollard Dr Richmond VA 23226	AN	<b>PETRES</b> , MD, Robt Evan Box 34, M C V Station Richmond VA 23298	OBG	<b>REDMAN</b> , MD, Richard Don 6722 Patterson Avenue Richmond VA 23226	PS
<b>MC GUIRE JR</b> , MD, Hunter H Mc Guire V A Hosp (112) Richmond VA 23249	GS	<b>MONROE</b> , MD, Paul S. 5855 Brems Rd, #208 Richmond VA 23226	GE	<b>NUARA</b> , MD, Jos Carl 2621 Grove Ave Richmond VA 23220	CD	<b>PETTY</b> , MD, Carroll Thomas 2006 Brems Rd, Ste 201 Richmond VA 23226	PS	<b>REDWINE</b> , MD, Fay Oneal Box 34, M C V Station Richmond VA 23298	OBG
<b>MC KAY</b> , MD, James 7103 B Jahnke Rd Richmond VA 23225	GP	<b>MONROE</b> , MD, Willys Moore 5303 Ditchley Rd Richmond VA 23226	CLP	<b>O BRIEN</b> , MD, John Patrick 118 Libby Ave Richmond VA 23226	IM	<b>PHILLIPS</b> , MD, Charles W. 7702 Parham Road Richmond VA 23229	CD	<b>REED</b> , MD, John Ernest 1508 Cedar Bluff Dr Richmond VA 23233	NM
<b>MC KEOWN</b> , MD, Chas E 110 Windsor Way Richmond VA 23221	OM	<b>MONTAGUE</b> , MD, David Lee 5855 Brems Rd Richmond VA 23226	OBG	<b>O'BANNON III</b> , MD, John Maurice 1805 Monument Avenue Richmond VA 23220	N	<b>PHILLIPS</b> , MD, Robt M Box 340 Chester VA 23831	FP	<b>REED</b> , MD, Wellford C 4501 Bromley Lane Richmond VA 23221	CD
<b>MC MULLAN</b> , MD, Francis H 1812 Monument Avenue Richmond VA 23220	D	<b>MONTAGUE</b> , MD, John W 109 Windsor Way Richmond VA 23221	OTO	<b>OATES III</b> , MD, James Franklin Medical Science Center Richmond VA 23226	GS	<b>PILCHER JR</b> , MD, Robert M 7135 Jahnke Rd Richmond VA 23225	ORS	<b>REESE</b> , MD, Geo Walter 8906 Gingerway Drive Richmond VA 23229	IM
<b>MC NEER</b> , MD, Paul Randolph 204 N Hamilton St Richmond VA 23221	OPH	<b>MONTEIRO</b> , MD, Idelonso C 402 September Drive Richmond VA 23229	AN	<b>OGLESBY SR</b> , MD, F Elliott 3500 Grove Ave Richmond VA 23221	GP	<b>PILLSBURY</b> , MD, Susan Lewis 9301 Hull Street Road Richmond VA 23236	PD	<b>REESE</b> , MD, Mitchell S. 5801 Brems Road Richmond VA 23226	R
<b>MC GEE JR</b> , MD, Francis E 1805 Monument Avenue Richmond, VA 23220	N	<b>MOORE II</b> , MD, Frederic Potts 3602 Monument Ave Richmond VA 23230	PD	<b>OLIFF</b> , MD, Geo Anthony 1509 Jonquill Dr Richmond VA 23233	DR	<b>PINSKER</b> , MD, M. Craig 10412 Falconbridge Drive Richmond VA 23233	AN	<b>REGAN</b> , MD, Wm Whitfield 900 North Hamilton Street Richmond VA 23221	GE
<b>MC GLYNN</b> , MD, Fred J. 8919 Three Chopt Road Richmond VA 23229	ORS	<b>MOORE III</b> , MD, D. Parker 1613 Florence Avenue Chester, VA 23831	FP	<b>OLSHANSKY</b> , MD, Kenneth 7929 Chowning Cr Richmond VA 23229	PS	<b>PIZZANI</b> , MD, Eddy 5855 Brems Rd, #302 Richmond VA 23226	IM	<b>REGELSON</b> , MD, William Box 273, M C V Station Richmond VA 23298	ND
<b>MC GROOM</b> , MD, David John 5855 Brems Road, #302 Richmond VA 23226	IM	<b>MOORE</b> , MD, Robert Patrick 7 Rose Hill Road Richmond VA 23229	IM	<b>OPPENHEIMER</b> , MD, Wm Mayo 7601 Forest Ave Suite 114 Richmond VA 23229	OBG	<b>POLE</b> , MD, Frank N 2002 Brems Rd Ste 202 Richmond VA 23226	U	<b>REIN</b> , MD, Walter J 5 Glenbrooke Circle East Richmond VA 23229	OPH
<b>MC NEER</b> , MD, Keith Wilson 5700 W Grace Street, #110 Richmond VA 23226	OPH	<b>MOREY</b> , MD, Dennis A J 900 North Hamilton Street Richmond VA 23221	GE	<b>OUTEN</b> , MD, Carl D. 701 North Courthouse Rd Richmond VA 23236	OPH	<b>POLIN</b> , MD, Gerald Mark 9805 Drovon Drive Richmond VA 23233	CHP	<b>RENNIE</b> , MD, Laurie Earl 1805 Monument Ave Richmond VA 23220	N
<b>MC TAMANEY</b> , MD, James Paul 1400 Westwood Ave, #101 Richmond VA 23227	GS	<b>MORGAN JR</b> , MD, Carlisle Lee 1602 Skipwith Road Richmond VA 23229	DR	<b>OVERTON</b> , MD, Thos P 7000 Patterson Ave Richmond VA 23226	PD	<b>POLLACK JR</b> , MD, David 5100 Monument Ave, Apt 403 Richmond VA 23230	IM	<b>REPASS</b> , MD, James Albert 7702 Parham Road Richmond VA 23229	IM
<b>MEADOR</b> , MD, Blake W 4105 Sulgrave Road Richmond VA 23221	PUD	<b>MORRISSETT</b> , MD, Leslie E 5104 Riverside Dr Richmond VA 23225	LAR	<b>OWEN JR</b> , MD, Duncan Shaw Box 647, M C V Station Richmond VA 23298	RHU	<b>POLLOCK JR</b> , MD, Chas Allen 5700 Old Richmond Ave Ste 2 Richmond VA 23226	IM	<b>REYNOLDS</b> , MD, Richard R. 7107 Jahnke Road Richmond VA 23225	CDS
<b>MECKS</b> , MD, Charles H 4202 E Oxford Circle Richmond VA 23221	AN	<b>MORRISSETTE III</b> , MD, Wm P. Box 446 Midlothian, VA 23113	FP	<b>OWEN JR</b> , MD, Fletcher Bailey 1407 Cummings Dr Richmond VA 23220	OS	<b>POPE JR</b> , MD, John Henry 7127 Jahnke Rd Richmond VA 23225	US	<b>RICE JR</b> , MD, Marion Lee 3801 Patterson Avenue Richmond VA 23221	GE
<b>MECKS</b> , MD, Thomas George 204 North Hamilton Street Richmond, VA 23221	OTO	<b>MORRISSETTE</b> , MD, W. P. Box 446 Midlothian, VA 23113	FP	<b>OWEN JR</b> , MD, Heth 608 Lee Med Bldg Richmond VA 23220	AN	<b>PORTER JR</b> , MD, Geo Wm 7133 Jahnke Rd Richmond VA 23225	OM	<b>RICHARDS JR</b> , MD, Chas N 7125 Jahnke Rd Richmond VA 23225	GP
<b>MEHRHOF JR</b> , MD, Austin I Box 154, M C V Station Richmond VA 23298	PS	<b>MUELLER</b> , MD, Dawn Grigg Box 276, M C V Station Richmond VA 23298	PD	<b>OWEN</b> , MD, John Thos 211 Averbury Road Richmond VA 23235	FP	<b>PORTER</b> , MD, Reno Russell Box 12, M C V Station Richmond VA 23298	CD	<b>RICHARDS</b> , MD, Nelson G 1453 Johnston-Willis Dr Richmond VA 23235	N
<b>MEILLER</b> , MD, Joan Mason 2002 Brems Rd Ste 200 Richmond VA 23226	CHP	<b>MUELLER</b> , MD, John M 2002 Brems Rd, Lower Level Richmond VA 23226	US	<b>OWENS JR</b> , MD, Maurice B 1013 W Franklin St Richmond VA 23220	A	<b>POULOS</b> , MD, Nicholas Geo 3536 Grove Ave Richmond VA 23221	GS	<b>RICHARDSON</b> , MD, Herman M Midlothian VA 23113	GP
<b>MELLETTTE</b> , MD, M Susan Jackson Box 207, M C V Station Richmond VA 23298	IM	<b>MULLER</b> , MD, John Garrett 701 West Grace Street Richmond, VA 23220	GP	<b>OWNBY JR</b> , MD, Ralph 2924 Brook Rd Richmond VA 23220	PD	<b>PRICE</b> , MD, Robt Lee 4001 Croatan Rd Richmond VA 23235	PH	<b>RIDDICK</b> , MD, David Haydon 2002 Brems Road Lower Level Richmond VA 23226	IM

\* = AMA membership

RILEY, MD, Chas R 3604 Monument Ave Richmond VA 23230	GS	SALZBERG, MD, Arnold Martin Box 15, M C V Station Richmond VA 23298	GS	SLAGEL, MD, Dale Everett 330 South 4th Street Richmond, VA 23219	IM	STRACHAN, MD, Michael Joel 2002 Breomo Road Richmond, VA 23226	IM	TORRES-LISBOA, MD, Patricia 3001 Fifth Avenue Richmond, VA 23222	P
RILEY, MD, P. Thomas P O Box 446 Midlothian, VA 23113	FP	SANDERS, MD, Donald Powell 204 N Hamilton Street Richmond, VA 23221	FP	SMITH JR., MD, William Kyle 5801 Breomo Road Richmond VA 23226	CD	STRATFORD, MD, Thos Peirson 201 N Hamilton St Richmond VA 23221	OPH	TOSI, MD, Richard Englund 325 Oak Lane Richmond, VA 23226	IM
RITCHIE JR., MD, Geo G 102 Durrington Circle Richmond, VA 23236	P	SASSER JR., MD, Frank M 1407 Cummings Drive Richmond VA 23220	OM	SMITH, MD, Alfred L 100 Matoaka Rd Richmond VA 23226	IM	STREICKER, MD, Wm F 2200 Monument Avenue Richmond, VA 23220	EM	TRICE, MD, Clarry Clyde 211 Berkshire Richmond VA 23221	US
RIVADENEIRA, MD, Colon 6415 Mechanicsville Tr Pk Mechanicsville, VA 23111	PD	SAUNDERS, MD, John Rudolph 1500 Westbrook Ave Richmond VA 23227	P	SMITH, MD, Crawford C 10627 Harborough Place Richmond, VA 23233	CRS	STRINGER, MD, Kenneth Robert 500 Pocoshock Place Richmond VA 23235	D	TRICE, MD, Ernest Randolph 7702 Parham Road Richmond VA 23229	DR
RIVERS, MD, Cullen B 7103-C Jahneke Road Richmond VA 23225	IM	SAVAGE, MD, Bernard M P O Box 26783 Richmond VA 23261	R	SMITH, MD, J Earle 3020 Kenbury Rd Richmond VA 23235	GP	STROTHER, MD, Arnold F 406 September Dr Richmond VA 23229	CHP	TRICE, MD, Robt Palmer 3500 Kensington Ave Suite # 10 Richmond VA 23221	GP
ROBERTS, MD, Bruce Taylor P O Box 5306 Richmond VA 23226	IM	SAYLOR, MD, Edward Michael 5 West Nine Mile Road Highland Springs, VA 23075	EM	SMITH, MD, James Alexander 2956 Hathaway Rd, Tower 702 Richmond VA 23225	PH	STROUBE, MD, Robt Bruce 109 Governors' St Richmond, VA 23219	PH	TROLAND, MD, Chas Edward 3800 Patterson Ave Richmond VA 23221	NS
ROBERTSON JR., MD, Giles M 900 N Hamilton St Richmond VA 23221	GE	SCAVULLO, MD, Blaise 4103 Hickory Road Richmond, VA 23235	P	SMITH, MD, Leroy 1805 Monument Ave Richmond VA 23220	PS	STUART, MD, Wm Thos 7702 Parham Rd Richmond VA 23229	U	TRUESDELL, MD, Frank B 42 Chatham Sq Richmond VA 23226	OS
ROBERTSON JR., MD, Charles H 5855 Breomo Rd, Ste 509 Richmond VA 23226	IM	SCHERER, MD, Edward Underwood 6423 Handy Lane Richmond, VA 23226	EM	SMITH, MD, Lindley Theodore 112 East Clay St Richmond VA 23219	OPH	SUTER, MD, Cary Grayson Box 599, M C V Station Richmond VA 23298	N	TUCKER, MD, Stanley Cole 7702 Parham Road Richmond VA 23229	CD
ROBERTSON, MD, Elmer S 700 W Grace Street Richmond VA 23220	CD	SCHIEKEN, MD, Richard M P O Box 26 Richmond, VA 23298	PDC	SMITH, MD, Mason 2035 Monument Ave Richmond VA 23220	OPH	SUTPHIN, MD, Adney Kemple 1805 Monument Ave Richmond VA 23220	OM	TUCKER, MD, Weir M 6208 Topoan Place Richmond VA 23226	N
ROBERTSON, MD, Louise Wilkes Box 223, M C V Station Richmond VA 23298	PDC	SCHIFF, MD, Graunum Robt 9603 Whitmore Drive Richmond VA 23229	P	SMITH, MD, Maurice J Vernon Box 118, M C V Station Richmond VA 23298	U	SUTTON IV., MD, Valvin Earl 10 E Leigh Street Richmond VA 23219	IM	TUCKER, MD, Wm T 7601 Forest Ave Richmond VA 23229	IM
ROBERTSON, MD, William A 5500 Monument Ave Ste L Richmond VA 23226	TS	SCHLOBOHM, MD, Philip G. P O Box 9127 Richmond, VA 23227	P	SMITH, MD, Maynard Putney 1835 Monument Ave Richmond VA 23220	OTO	SUTTON, MD, Chas Edward 10 E Leigh St Richmond VA 23219	GP	TULO, MD, Nicolas Paul 2012 Monument Avenue Richmond VA 23220	IM
ROBINS JR., MD, Spotswood 207 Matoaka Road Richmond VA 23226	GYN	SCHWARZ, MD, Maurice Chaskiel 1400 Westwood Ave, #306 Richmond VA 23227	HEM	SMITH, MD, Ray Huey P O Box 13470, #D Richmond VA 23225	CD	SVOBODA, MD, Jos Robt Med Science Bldg Richmond VA 23226	OPH	TUNNER, MD, Wm Sams 5855 Breomo Rd, #204 Richmond VA 23226	U
ROBINSON III, MD, Grover C. 9603 Gayton Road Richmond, VA 23233	PD	SCOGGINS, MD, Robt Bruce 1820 Monument Ave Richmond VA 23220	D	SMITH, MD, Rodney Hall 1429 Johnston Willis Dr Richmond VA 23235	PUD	SWEET, MD, Raymond Charles 1888 S. Sycamore Street Petersburg, VA 23805	N	TURNER, MD, Mary Ann Box 615 Richmond VA 23298	DR
ROBINSON, MD, Frederick Danl 11800 Heathmere Cres Midlothian VA 23113	CD	SCOTT, MD, Robt Bradley Box 214, M C V Station Richmond VA 23298	IM	SMITH, MD, Thomas Adrian 8921 Three Chopt Road Richmond VA 23229	N	SYLVEST, MD, Vernon Martin 1516 Ednam Forest Dr Richmond VA 23233	PTH	TYLER, MD, Gilman Rackley 4304 Cambridge Road Richmond VA 23221	CD
ROBINSON, MD, James F 1810 Monument Ave Richmond, VA 23220	D	SEEMAN, MD, Irvin Jay 5855 Breomo Road, #208 Richmond, VA 23226	IM	SMITH, MD, Wade Kilgore Box 162, M C V Station Richmond VA 23298	HEM	TALLEY III, MD, Danl Doak 120 S Wilton Rd Richmond VA 23226	R	ULMER, MD, Jack L 8910 Tolman Road Richmond VA 23229	NS
ROBINSON, MD, Wm Mayer 3026 W Cary St Richmond VA 23221	GP	SEITZ, MD, Donald G 7601 Forest Ave, #228 Richmond VA 23229	ORS	SNEAD JR., MD, Lawrence O 1104 W Franklin St Richmond VA 23220	R	TALMAN, MD, Edward Armistead 24 Tapoan Rd Richmond VA 23226	GS	VAUGHAN, MD, David Du Puy 7702 Parham Rd Richmond VA 23229	IM
ROGERS, MD, John Fredric 4908 Riverside Drive Richmond, VA 23225	PD	SELHORST, MD, John B Box 599 Richmond VA 23298	N	SNEAD, MD, Ronald Wilson Va Trtment Cen Children, Box 1 Richmond VA 23201	CHP	TANNER, MD, W Woodrow Rt. 2, Box 159K Powhatan, VA 23139	AN	VELO, MD, Anthony Gomez 1407 Johnston Willis Dr Richmond VA 23235	NS
ROMAINE, MD, Chas Nichols 5700 W Grace St Richmond VA 23227	OPH	SELLMAN, MD, James E 1500 Westbrook Avenue Richmond, VA 23227	CHP	SNODDY, MD, John Wm. 1400 Westwood Ave, #101 Richmond VA 23227	GS	TATTERSALL, MD, Mary Agnes Ruthville VA 23147	IM	VENNART, MD, Geo P 1200 E Broad St Div Path Richmond VA 23298	PTH
ROONEY, MD, Mervyn Stuart C 5501 Cary St Rd Richmond VA 23226	AN	SELPH, MD, James Anderson 1203 Rothesay Circle Richmond VA 23236	U	SOBIESKI, MD, Thomas John 11943 Mountain Laurel Rd Richmond VA 23236	IM	TAYLOR, MD, Donald A. 1825 Monument Ave Richmond, VA 23220	N	VETROVEC, MD, Geo Wayne 5212 New Kent Road Richmond VA 23225	IM
ROPER, MD, Barry Edward 8132 Forest Hill Ave Richmond, VA 23235	OPH	SHAI, MD, Frad Thomas 2821 Parham Road, #204 Richmond VA 23229	OTO	SOLAN, MD, Stuart Miley 10431 Patterson Ave Richmond, VA 23233	FP	TAYLOR, MD, John Richard 13609 Kingsmill Road Midlothian, VA 23113	N	VILLAROMAN, MD, Ruben R 45-B Williamsburg Road Sandston VA 23150	IM
ROSANELLI JR., MD, Peter 5855 Breomo Rd Ste 301 Richmond VA 23226	OBG	SHANHOLTZ, MD, Mack Irvin 5912 Upham Drive Richmond VA 23227	PH	SOLOMON, MD, Stuart 2821 Parham Road Richmond VA 23229	PD	TEMPLE, MD, James Edmond 1443 Johnston Willis Dr Richmond VA 23235	AN	VILSECK JR., MD, Jos Richard 2000 Breomo Rd #204 Richmond VA 23226	A
ROSE JR., MD, Leslie Wm 5500 Monument Ave Richmond VA 23226	IM	SHARPE, MD, Alton Rivington Box 1, M C V Station Richmond VA 23298	NM	SORREL, MD, Karan Campbell 2230 Oakengate Lane Midlothian, VA 23113	PD	THEDIECK, MD, Charles Gerhard Parham Medical Center, Suite 2 Richmond, VA 23229	OBG	VINES, MD, Fredrick Sherwin Box 47, M C V Station Richmond VA 23298	DR
ROSENBERG, MD, Santord M. Box 34, M C V Station Richmond, VA 23298	OBG	SHAUGHNESSY, MD, Katherine T. 3536 Grove Avenue Richmond, VA 23221	OBG	SOUTH, MD, James Gregory 7702 Parham Road Richmond VA 23229	DR	THOMAS JR., MD, Harry 901 Hoaks Road Richmond, VA 23225	OBG	VINIK, MD, Melvin 9504 Carterwood Road Richmond VA 23229	R
ROWE, MD, Douglas Stephen 2004 Breomo Rd, #106 Richmond VA 23226	PS	SHAYNE, MD, Robt Stevan 4705 Buckingham Court Chester, VA 23831	PD	SOWERS, MD, Richard P. 4800 Pocahontas Rd Richmond, VA 23226	GP	THOMAS JR., MD, Harry R. P O Box 25206 Richmond, VA 23260	IM	VIOL, MD, Goffrey William Medical Science Center Libbie & Old Richmond Avas Richmond, VA 23226	IM
ROWLAND JR., MD, Harry S 7601 Forest Ave #337 Richmond VA 23229	U	SHEPHERD, MD, Eugene Bowie 3821 Seminary Ave Richmond VA 23227	IM	SPANIER, MD, Elliott J. 7149 Jahneke Road Richmond, VA 23225	P	THOMAS, MD, Carolyn E 11851 Wexwood Rd Richmond VA 23236	PTH	VITOLS, MD, Mintaus Mickey 1500 Westbrook Ave Richmond VA 23227	US
ROYAL, MD, Frank Spencer 1122 North Twenty-Fifth St Richmond VA 23223	GP	SHEPPARD, MD, L Benj 116 E Franklin St Richmond VA 23219	OPH	SPECTOR, DO, Paul M 500 N Boulevard Richmond VA 23220	OST	THOMAS, MD, George Walter 1401 Johnston Willis Dr Richmond VA 23235	PTH	VITSKY, MD, Maurice Sidney 3403 Wythe Ave Richmond VA 23221	OBG
ROYAL, MD, Harry Willis P O Box 26524 Richmond VA 23261	OBG	SHIELD JR., MD, James Asa 7149 Jahneke Road Richmond VA 23225	P	SPENCER, MD, Frederick J Box 212, M C V Station Richmond VA 23298	PH	THOMAS, MD, Pendleton Emmett 5700 Old Richmond Rd Richmond VA 23226	GYN	VITSKY, MD, Meyer 11801 Bedfordshire Sq Richmond VA 23233	GYN
ROYSTER, MD, Henry Page 7129 Jahneke Rd Richmond VA 23225	GS	SHIM, MD, Chi Yun 11551 Rex Moor Dr Richmond VA 23235	AN	SPENCER, MD, Henry S 7702 Parham Road Richmond VA 23229	R	THOMPSON JR., MD, Wm T 4602 Sulgrave Rd Richmond VA 23221	IM	VLANCEVIC, MD, Z. Reno Box 711, M C V Station Richmond, VA 23298	GE
RUDDY, MD, Shaun Jos Box 263, M C V Station Richmond VA 23298	RHU	SHIVEL, MD, Glen L 3500 Kensington Ave Richmond, VA 23221	R	SPLANE III, MD, George Russell 1805 Monument Ave, #316 Richmond VA 23220	P	THORNTON, MD, John L 1401 Johnston Willis Dr Richmond VA 23235	PTH	VOKAC, MD, Vaclav Albart 1211 Sherwood Avenue Richmond, VA 23220	GE
RUZINSKI, MD, Dennis J. 2710 Newquay Lane Richmond VA 23236	AN	SHOWALTER, MD, Henry Bernard 206 Canterbury Road Richmond VA 23221	R	SPORN, MD, I Norman 1400 Westwood Ave, #302 Richmond VA 23227	IM	THORPE, MD, Alice V 5801 Breomo Rd Richmond VA 23226	CD	VRANIAN, MD, N Michael 7702 Parham Road Richmond VA 23229	IM
RUFFIN, MD, Herbert G 2508 Hilliard Rd Richmond VA 23228	GP	SILVERMAN, MD, Joel J Box 710, M C V Station Richmond VA 23298	P	STALKER, MD, Campbell Grieve 213 Gun Club Road Richmond VA 23221	CDs	THRIFT, MD, Geo N 303 Clovelly Rd Richmond VA 23221	OTO	WADDELL, MD, Marion Crockett Med Arts Bldg Richmond VA 23219	OPH
RUSHER, MD, Wm De Witt 3500 Kensington Ave Richmond VA 23221	OPH	SIMS, MD, Angela Valencia 5024 Sylvan Road Richmond, VA 23225	CHP	STARKMAN, MD, Martin T. 7702 Parham Road Richmond, VA 23229	NEP	THURMAN, MD, Wm Allen 5115 Cary St Rd Richmond VA 23226	DR	WALKER, MD, Barry Quentin 5801 Breomo Rd Richmond VA 23226	R
RUSSELL JR., MD, John A 513 Forest Avenue Richmond VA 23229	P	SINGER, MD, Robt Perry 1651 Parham Road Richmond VA 23229	NS	STENSLAND, MD, Mark S. 2912 Fox Chase Drive Midlothian, VA 23113	FP	TISNADO JR., MD, Jaime Box 615, M C V Station Richmond VA 23298	R	WARD, MD, Chas Harper 5 Raven Rock Ct Richmond VA 23229	EM
RYAN, MD, Arthur Eugene 2 Country Square Lane Richmond VA 23229	OM	SINGH, MD, Harinder Paut 3601 Dill Rd Richmond VA 23222	AN	STEWART, MD, Lauraine 3831 Old Gun Road-E Midlothian, VA 23113	PD	TITUS, MD, Cloyd Kent 14 Stonehurst Green Richmond VA 23226	IM	WARD, MD, Wm O Neil 2004 Breomo Road, #202 Richmond VA 23226	HYP
SAKOWSKI JR., MD, Anthony D 5855 Breomo Road, #508 Richmond VA 23226	OPH	SINGH, MD, Kanwal S. Box 631, M C V Station Richmond, VA 23298	NS	STEWART, MD, William Bruce 511 Medical Arts Building Richmond, VA 23219	CRS	TONEY, MD, Ronald W 418 Libbie Avenue Richmond VA 23226	FP	WARE III, MD, Harry Hudnall 5855 Breomo Rd Ste 205 Richmond VA 23226	OBG
SALOMONSKY, MD, Anita B. 1201 Loch Lomond Ct Richmond, VA 23221	AN	SISMANIS, MD, Aristides Box 146 Richmond, Va 23298	OTO	STONNINGTON, MD, Henry H Box 677, M C V Station Richmond, VA 23298	PM	TOONE JR., MD, Elam Cooksie 4402 West Franklin St Richmond VA 23221	RHU	WARE, MD, James Latane 5855 Breomo Rd Richmond VA 23226	PS

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WARKENTIN, MD, John R 2621 Grove Avenue Richmond, VA 23220	IM	WILLIAMS, MD, David C. 7901 Burrundie Drive Richmond, VA 23225	GP	ZAKAIB, MD, Edward Albert 7117 Jahnke Rd Richmond VA 23225	FP	BLAYLOCK, MD, Wm Mc Gehee 3259 Somerset St Sw Roanoke VA 24014	RHU	CREEKMORE, MD, Robt Sherman 2037 Crystal Springs Ave Roanoke VA 24014	DR
WARREN, MD, David L. P O Box 27003 Richmond, VA 23261	IM	WILLIAMS, MD, Geo Harry Box 146, M C V Station Richmond VA 23261	OTO	ZALIS III, MD, Oreste 203 Tarrytown Dr Richmond VA 23229	P	BOCKNER, MD, Andrew Chas 101 Mountain Avenue Roanoke VA 24016	P	CRICKENBERGER, MD, Dallas P 2110 Carolina Ave, Sw Roanoke VA 24014	ORS
WASHINGTON, MD, Thos Boyd 412 Libbie Avenue Richmond VA 23226	U	WILLIAMS, MD, Louis H 2750 Stratford Road Richmond VA 23225	OBG	ZAMBRANA, MD, Benj Franklin 4405 Forest Hill Avenue Richmond VA 23225	FP	BOGGESE, MD, H Preston 4403 Grandin Road, Sw Roanoke VA 24018	PD	CROCKETT JR., MD, Chas L 3136 Somerset St S W Roanoke VA 24014	HEM
WASSERMAN, MD, Albert J 1305 Lake Avenue Richmond VA 23226	PA	WILLIAMS, MD, Marvin Thomas 2837 Poyntelle Rd Richmond VA 23235	FP	ZANGA, MD, Jos Robt 9114 Dondra Drive Richmond VA 23229	PD	BONDURANT, MD, Robt F 2831 Stephenson Ave Sw Roanoke VA 24014	IM	CROW JR., MD, William Cecil P O Box 280 Fincastle, VA 24090	FP
WASSERMAN, MD, Brian Mark 7157 Jahnke Road Richmond VA 23225	IM	WILLIAMS, MD, Mason Miller 5855 Breimo Road, # 406 Richmond VA 23226	OTO	ZELENAK, MD, James Joseph 3027 Kenmore Road Richmond VA 23225	NM	BONO, MD, Jos Albert 1315 Second St Sw Roanoke VA 24016	GS	CROWGEY, MD, Junius Ellett P O Box 1789 Roanoke VA 24008	OPH
WATKINS, MD, Franklin P 4315 Grove Ave Richmond VA 23221	ORS	WILLIAMS, MD, Richard K 2015 Monument Ave Richmond VA 23220	OPH	ZFASS, MD, Hyman Saml 2502 Monument Ave Richmond VA 23220	IM	BOWLES, MD, Paul Elwood 1201 Third St Roanoke VA 24016	PD	CRUM, MD, Jerry Brice 1138 Second St Sw Roanoke VA 24016	OPH
WATLINGTON, MD, Charles O. Box 145, M C V Station Richmond, VA 23298	IM	WILLIAMS, MD, Robert K. 5004 Monument Ave Richmond, VA 23230	P	ZIMBERG, MD, Yale H 5855 Breimo Rd Suite 405 Richmond VA 23226	TS	BOYD JR., MD, John Otto Route #1, Box 215 Goodview, VA 24095	GER	CRUSER, MD, Fred S V A Hosp Salem VA 24153	P
WATSON, MD, T. Lepierre 4301 Meadowdale Blvd Richmond, VA 23234	GP	WILSON JR., MD, Henry H 1409 Johnston Willis Dr Richmond VA 23235	PS	ROANOKE					
WAYBRIGHT, MD, Edward A 2917 Kenmore Road Richmond, VA 23225	N	WILSON, MD, Cleude Watson 1651 Parham Road Richmond VA 23229	NS	AIZCORBE, MD, Oscar Ramiro Va Hospital Salem, VA 24153	PM	BRAY JR., MD, Charles B 1240 3rd St S W Roanoke VA 24016	ORS	CUESTA, MD, Maximo Lopez Roanoke Memorial Hospital Roanoke VA 24033	OBG
WEAVER, MD, John A. 1000 N Thompson St Richmond, VA 23220	DR	WILSON, MD, Jeffrey K. 2222 Monument Avenue Richmond, VA 23220	ORS	ALDEA, MD, Erlinda Doncello Route 2, Box 396 Covington VA 24426	PD	BROBST, MD, Henry Thos 2037 Crystal Spring Ave Roanoke VA 24014	PS	CUTTER, MD, Edgar Burford 237 Franklin Rd Sw Roanoke VA 24016	GS
WEAVER, MD, Yvonne J. 1000 N Thompson St Richmond, VA 23230	CD	WINGFIELD, MD, Wm Lynn Route 4, Box 160 Ashland VA 23005	IM	ALEXANDER III, MD, H C 2037 Crystal Spring Ave S W Roanoke VA 24014	IM	BROCHU, MD, Francis Louis Vet Admin Hosp Salem VA 24153	ABS	DAVIDSON III, MD, Jesse T. 1234 Franklin Road, Sw Roanoke, VA 24016	GS
WEDD III, MD, George 4301 Meadowdale Blvd Richmond, VA 23234	FP	WINKLER, MD, Chas Pinckney 3500 Kensington Ave Richmond VA 23221	R	ALLEN JR., MD, Robert W. Route 4, Box 288 Troutville, VA 24175	PD	BRUBAKER, MD, Herman W 5501 Williamson Rd Sw Roanoke VA 24012	FP	DAVIS, MD, Algernon Gibson 3922 Electric Rd Sw Roanoke VA 24018	
WEGER, MD, Marvin Louis Parham Med Bldg 8600 Ouicocasin Richmond VA 23229	FP	WINN, MD, Washington Carlyle 3500 Grove Ave Richmond VA 23221	OBG	ALLEN, MD, John Thomas 1234 Franklin Road, Sw Roanoke, Virginia 24016	U	BUMGARDNER JR., MD, Jack H 209 Maple Ave Rocky Mount VA 24151	FP	DAVIS, MD, Frederick Ferdon 820 King James St Sw-Apt B Roanoke VA 24014	OS
WEIMER, MD, Geo A 7702 Parham Rd Richmond VA 23229	AN	WISE JR., MD, James Lafayette Rt 1 Box 137 Charles City VA 23030	GP	AMOS, MD, Jesse Francis 209 Maple Ave Rocky Mount VA 24151	FP	BURCH, MD, John Gordon 2601 Franklin Road, Sw Roanoke, VA 24014	N	DAVIS, MD, Wm Vaughan 1215 3rd Street, Sw Roanoke, VA 24016	OBG
WEINBERG, MD, Robert Stephen Box 262, M C V Station Richmond, VA 23298	OPH	WITTKAMP, MD, Bernard F. 8710 Choctaw Road Richmond, VA 23235	IM	ATKINSON, MD, Leigh Oliver 1900 Electric Rd Salem VA 24153	AN	BURNSTEIN, MD, Alan V. 2037 Crystal Spring Ave, Sw Roanoke, VA 24014	GE	DE BECK, MD, Thos Wede 2601 Franklin Rd Sw Roanoke VA 24014	N
WEINSTEIN, MD, Julian 5204 Patterson Ave Richmond VA 23226	PD	WOOD, MD, Maurice Box 251, M C V Station Richmond, VA 23298	FP	AUSTIN, MD, Joseph Lee 1615 Franklin Road, Sw Roanoke, VA 24016	CD	BURTON, MD, Calvin Thos 209 Medical Arts Bldg Roanoke VA 24011	OPH	DE VERTER, MD, John Scott 1902 Braeburn Dr Salem VA 24153	P
WEISGER III, MD, Benj B 8921 Gingerway Drive Richmond VA 23229	GE	WOOD, MD, Robt L 5728 Hillview Dr Mechanicsville VA 23111	PH	AYILIDZ JR., MD, Vedii 511 Boulevard Ave Salem VA 24153	GS	BUTLER III, MD, William W 1234 Franklin Rd S W Roanoke VA 24016	U	DICKERSON, MD, Shelby Clark P O Box 13367 Roanoke VA 24033	IM
WELLS, MD, James Morgan 6823 Kensington Ave Richmond VA 23226	PD	WOODHOUSE III, MD, Robt W 814 Baldwin Rd Richmond VA 23229	OM	BAILEY, MD, Dewey James 2037 Crystal Spring Ave Sw Roanoke VA 24014	IM	CANNON, MD, M. Marci Route 3, Box 408 Salem, VA 24153	IM	DICKINSON, MD, Clara R King 215 Hershberger Nw #F-1B Roanoke VA 24012	OS
WENLEDER, MD, Rudolf Bernard P O Box 13019 Richmond VA 23225	AN	WOODLIEF, MD, Ray Marshall 3724 Falstone Rd Richmond VA 23234	DR	BALL, MD, Lawrence Carter 3912 Winding Way Rd Sw Roanoke VA 24015	PH	CARMICHAEL, MD, Elizabeth B. 204 S Maple Street Vinton, VA 24139	FP	DILL, MD, James Ellis 1310 Third Street Roanoke, VA 24016	IM
WETCHLER, MD, Stewart J. Box 34, M C V Station Richmond, VA 23298	OBG	WOOTTON, MD, Jane P 17 Tapoon Rd Richmond VA 23226	IM	BALLENGER, MD, Fred Jackson 1117 South Jefferson St Roanoke, VA 24016	IM	CARMICHAEL, MD, Paul A. 4229 Colonial Ave, Bldg D Roanoke, VA 24018	GS	DILLON, MD, Ronald Williams 400 Burwell Street Salem, VA 24153	OPH
WHEELER, MD, William Edge 5801 Breimo Road Richmond, VA 23226	R	WOOTTON, MD, Percy 7601 Forest Ave, #340 Richmond VA 23229	CD	BARNHART JR., MD, Ruth 3533 Penarth Rd Sw Roanoke VA 24014	PD	CASSADA, MD, Wm Abraham 4513 Laurelwood Dr Sw Roanoke VA 24018	R	DONATO, MD, Antonio Tueson 1125 S. Jefferson Street Roanoke, VA 24016	GS
WHIPPLE, MD, Terry Lane 8919 Three Chopt Rd Richmond, VA 23229	ORS	WORLAND, MD, Richard L 2911 Grove Ave Richmond VA 23221	ORS	BARRETT, MD, Christine E. 312 North Main Street Rocky Mount, VA 24151	GS	CASPERN, MD, Louis J. 2913 Lockridge Road, Sw Roanoke, VA 24014	EM	DONNELLY, MD, Thos Edward 1315 2nd St Sw Roanoke VA 24016	IM
WHITAKER, MD, Joyce Lafon 8325 Brookfield Road Richmond, VA 23227	PD	WRIGHT, MD, R Lewis 4908 Monument Ave Richmond VA 23230	NS	BARTLEY JR., MD, Homer 216 Boxley Bldg Roanoke VA 24011	GP	CASTLE, MD, James Richard 1111 S. Jefferson Street Roanoke, VA 24016	IM	DOSS, MD, Julien Booth 2309 Carter Rd Sw Roanoke VA 24015	
WHITE, MD, William Richard 1651 Parham Road Richmond, VA 23229	NS	WYATT, MD, Davis Burton Parham Prof Park 2801 Parham Road Richmond VA 23229	PD	BASILE, MD, Michael Joseph 1310 Third Street, S W Roanoke, VA 24016	IM	CHAKRAVORTY, MD, Ranec C. V A Hosp Salem, VA 24153	GS	DRIVER, MD, Seml Francis 3426 W Ridge Circle, Sw Roanoke VA 24014	P
WHITLEY, MD, Donald Phillip 98 Old Bridge Lane Richmond VA 23229	R	WYSOR, MD, Edwin Snead Mechanicsville VA 23111	GP	BASILE, MD, Vincent T 4330 Old Cve Spring Rd Sw Roanoke VA 24018	AN	CHAMBERLAIN, MD, Richard R 1234 Franklin Rd Roanoke VA 24016	GYN	DUCEY, MD, Kevin Francis 2037 Crystal Spring Ave, Sw Roanoke, VA 24014	CDS
WICKHAM, MD, James Robt 2201 Grove Ave Richmond VA 23220	IM	YANCEY, MD, Henry A 1400 Westwood Ave Richmond VA 23227	ORS	BATCHELOR, MD, Geo Henry 1315 2nd Street, Sw, #304 Roanoke VA 24016	OTO	CLAGUE, MD, Allen Manville 1802 Braeburn Dr Salem VA 24153	GP	DUCKWALL, MD, Francis Jos 1802 Breeburn Dr Selem VA 24153	PD
WICKING, MD, David Kerndt 9 N 14th St Richmond VA 23219	FOP	YOUNG, MD, Young Ok 5801 Breimo Rd St Merys Hosp Richmond VA 23226	AN	BEAR JR., MD, Jos Wolte Box 566 Roanoke VA 24003	GP	CLAPSADDLE, MD, Gene Edward Route 2, Box 288 Moneta, VA 24121	FP	DUCKWORTH, MD, Elizabeth H. 3342 Dawn Circle, Sw Roanoke, VA 24018	AN
WIESINGER, MD, Herbert 2015 Monument Ave Richmond VA 23220	OPH	YOUNG, MD, Adrienna M. 9217 Groundhog Drive Richmond, VA 23235	IM	BEAVERS JR., MD, Aaron L. 5501 Williamson Rd Nw Roanoke VA 24012	FP	CLARK JR., MD, Wm Edwin 2037 Crystal Springs Ave Roanoke VA 24014	U	DUDLEY, MD, Frank Humbert P O Box 98 Gledhill VA 24092	GP
WIGAND, MD, James Peter 3705 Commodore Point Ct Midlothian, VA 23113	IM	YOUNG, MD, Harold Francis Box 631, M C V Station Richmond VA 23298	NS	BEAZLEY III, MD, Luthur A. 6141 Flamingo Drive Roanoke, VA 24018	PD	CLARK, MD, James Howard 2115 Crystal Spring Ave Roanoke VA 24014	CDS	DURHAM, MD, Alfred A. 2110 Caroline Ave Roanoke, VA 24014	ORS
WIGGINS, MD, Raymond Michael 9506 Old House Drive Richmond, VA 23233	OBG	YOUNG, MD, Reuben Barnes Box 65, M C V Station Richmond VA 23298	PD	BELL, MD, Houston Leshner 1315 Belle Aire Cir, Sw Roanoke VA 24018	OTO	CLARKE JR., MD, Eugene Joseph P O Box 12926 Roanoke VA 24029	GPM	EDDINS, MD, Wm Geo 2333 Stillion Cir Sw Roanoke VA 24018	GP
WILEY JR., MD, Edward James 7000 Patterson Ave Richmond VA 23226	PD	YOUNG, MD, W. Frederick 14005 Steepleston Dr Midlothian, VA 23113	P	BERRY, MD, Robt Edward Box 13367 Roanoke VA 24033	GS	CLARKSON, MD, Wm David 1906 Braeburn Drive Salem VA 24153	P	EDLON, MD, Guido Artes 4858 Glenbrook Dr Sw Roanoke VA 24018	AN
WILKERSON, MD, Vivian Myrtle Kenner Army Hosp Fort Lee VA 23801	PD	YOUNT JR., MD, B. Gerald 1807 Grove Avenue Richmond VA 23220	R	BERTHOLF, MD, Max Erwin Wendover Al U.S. 220 Deleville, VA 24083	FP	CLOUGH, MD, Lewis R 1884 Oak Drive Salem, VA 24153	GS	EDMONDS, MD, Robt Wm 5934 Castle Rock Road, Sw Roanoke VA 24018	IM
WILKES, MD, William Lee 1400 Westwood Ave, #307 Richmond VA 23227	OTO	ZACHARIAS, MD, Chas M 2621 Grove Avenue Richmond VA 23220	IM	BIVENS JR., MD, Carl Hill 213 McClanahan St Sw Roanoke VA 24014	END	COFFEY, MD, E. L. Box 297 Buchanan VA 24066	FP	EDMUNDS, MD, Keith Cestleton 1802 Breeburn Dr Salem VA 24153	FP
WILKINS, MD, Wm Thos 1407 Cummings Dr Richmond VA 23220	IM	ZACHARIAS, MD, Lawrence C 4312 Grove Ave Richmond VA 23221	IM	BIVINS, MD, Don Howard 2601 Franklin Rd, Sw Roanoke, VA 24014	N	COLE JR., MD, John P P O Box 8306 Roanoke VA 24014	OTO	EDWARDS III, MD, Richerd Thos 1603 Frenklin Rd Sw Roanoke VA 24016	IM
WILLIAMS, MD, Armistead M 204 N Hamilton St Suite 1 Richmond VA 23221	GS	ZACHARY, MD, John S. Box 549 Midlothian, VA 23113	PD	BLACKWELL, MD, James Edward 2037 Crystal Spring Ave Roanoke, VA 24014	R	COMER JR., MD, James Edward 127 Mc Clanehan Ave S W Roanoke VA 24014	D	ELIAS, MD, Wm Slimen 2601 Frenklin Rd Sw Roanoke VA 24014	P
WILLIAMS, MD, Chas Lee 4814 Riverside Dr Richmond VA 23225	GP	ZAIDMAN, MD, Gerald W. 809 N Tilden St Richmond, VA 23221	OPH	BLAIR, MD, Walter Bernard 1902 Breeburn Dr Salem VA 24153	P	CONWAY, MD, Kevin Box 8163 Roanoke VA 24014	AN	ELLETT JR., MD, Rutus P Route 3, Box 455 Moneta, VA 24121	OBG

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## 28 ROANOKE

ERDİM, MD, Feyyaz 3235 Somerset St S W Roanoke VA 24014	AN	HAMIDI-TOOSI, MD, Shakur 3242 Bromley Street Roanoke, VA 24018	OPH	KALVIS, MD, Elena Astra 3827 View Avenue Roanoke, VA 24018	PUD	MITCHELL, MD, Walton F 224 Main St New Castle VA 24127	GP	RICHARDS JR, MD, Lewis G Rd 1, Box 239-A Hardy, VA 24101	GS
ERWIN, MD, Wm Swadley 2037 Crystal Spring Ave Roanoke VA 24014	IM	HANABURY JR, MD, Mark R. P O Box 1789 Roanoke, VA 24008	OTO	KANG, MD, Young Sup 2037 Crystal Springs Ave S W Roanoke VA 24014	PS	MOIR, MD, Wm Maryon 2121 Rosalind Ave Roanoke VA 24014	IM	RIDGWAY-HULL, MD, Duvah B 1506 Franklin Rd S W Roanoke VA 24016	OBG
FAGAN, MD, Esther G 528 Woods Ave, Sw Roanoke, VA 24016	PH	HANCOCK, MD, John Dennis 1121 South Jefferson St Roanoke, VA 24016	OBG	KAUFMAN, MD, John Pearse 127 Mcclanahan St Roanoke VA 24014	D	MONAHAN, MD, Lawrence Keith 1111 South Jefferson St Roanoke VA 24016	IM	RIPLEY, MD, Louis Paul 1240 3rd St S W Roanoke VA 24016	ORS
FALKINBURG, MD, Newell R 1117 South Jefferson St Roanoke VA 24016	NEP	HARMS, MD, Carl Barnard 212 Highland Avenue Roanoke, VA 24016	OBG	KAUFMAN, MD, Wm H 16535 Yeoho Rd Sparks Glencoe MD 21152	D	MOORE, MD, Michael Judson 5005 Hunting Hills Dr Sw Roanoke VA 24016	IM	ROLLER, MD, Gerald Wm 1310 3D St S W Roanoke VA 24016	IM
FEAR, MD, Douglas D 707 S Jefferson, #303 Roanoke, VA 24011	GS	HARPOLE, MD, David H 204 Mc Clanahan St Roanoke VA 24014	TS	KEELEY, MD, Robert L 1234 Franklin Rd Roanoke VA 24016	TS	MORGAN, MD, John Edward 2037 Crystal Spring Ave Roanoke VA 24014	GE	ROMAN, MD, Jorge 1117 South Jefferson St Roanoke VA 24016	NEP
FERRY JR, MD, Darwin John 2601 Franklin Rd Sw Roanoke VA 24014	NS	HARRIS, MD, Ronald B 3419 Winterbury Lane Sw Roanoke VA 24014	OPH	KEFFER JR, MD, Ernest J 707 Med Arts Bldg Roanoke VA 24011	ORS	MORRIS III, MD, James Culvin 2233 Sanford Ave S W Roanoke VA 24014	PS	ROSENOFF, MD, Stephen Howard 2013 South Jefferson Street Roanoke VA 24014	ON
FISHER, MD, Richard Harding 1802 Braeburn Dr Salem VA 24153	ORS	HATCHER, MD, Wm F 2654 Robin Hood Road Se Roanoke VA 24014	OPH	KESSLER II, MD, A. Reit 1802 Braeburn Drive Salem, VA 24153	GS	MURRAY, MD, Robt Louis 4825 Buckhorn Road, Sw Roanoke VA 24014	R	ROTH, MD, Robt Frank Lewis Gele Clinic Salem VA 24153	PS
FORD, MD, George W 3835 Bosworth Dr, Sw Roanoke, VA 24014	OM	HAUSER, MD, J Bruce 2037 Crystal Spring Ave Roanoke, VA 24014	R	KEYS, MD, David Nilson P. O. Box 1789 Roanoke VA 24008	OTO	MYERS, MD, Alonzo H 5064 Crossbow Circle Roanoke, VA 24014	ORS	ROUTH, MD, William D. 1111 South Jefferson St Roanoke, VA 24016	IM
FOSTER, MD, Wm Leicester 2701 Melrose Ave N W Roanoke VA 24017	GP	HEFNER, MD, Charles A 213 Mcclanahan St, #206 Roanoke VA 24014	IM	KIM, MD, Young U 3604 Morning Dove Rd Sw Roanoke VA 24016	EM	NEWTON, MD, Richard Milton 2129 Rosalind Ave Sw Roanoke VA 24014	CD	ROYSTER JR, MD, Randolph Roanoke Mem Hospitals Roanoke, VA 24033	TR
FRANTZ, MD, Paul T 2037 Crystal Spring Ave, Sw Roanoke, VA 24014	CDS	HELVESTINE JR, MD, Frank Rt 4, Box 59 Roanoke VA 24018	U	KISTLER, MD, Philip Crosby 1234 Franklin Rd S W Roanoke VA 24016	TS	NIEDERLEHNER, MD, James R. 3320 Dawn Circle, S W Roanoke, VA 24018	AN	RUTH, MD, Gerald Jay 1215 3rd St Sw Roanoke VA 24016	GYN
FRAZIER, MD, Arthur Benj 3209 W Ridge Rd, Sw Roanoke, VA 24014	TR	HENNING, MD, George D 1240 3rd St S W Roanoke VA 24016	ORS	KNOFF, MD, Reuben De Loach 2037 Crystal Spring Ave S W Roanoke VA 24014	DR	NOLAN, MD, Donel Barry 2601 Franklin Rd Sw Roanoke VA 24014	N	RUTHERFORD JR, MD, William 2129 Rosalind Avenue Roanoke, VA 24014	CD
FRAZIER, MD, John Richard 1902 Braeburn Dr Salem VA 24153	CHP	HENRETTA, MD, Thomas Ross 1234 Franklin Rd Sw Roanoke VA 24016	GS	KRELL, MD, Linda Sue P O Box 13367 Roanoke, VA 24033	PD	NOLAND JR, MD, Eugene B 2632 Richelieu Avenue Roanoke, VA 24014	IM	SARVAY JR, MD, Thos Long 1315 Second Street Sw Roanoke VA 24016	P
FREEMAN, MD, Bruce G 1021 Third Street, Sw Roanoke, VA 24016	PS	HICKAM, MD, George Lindsay 1119 S Jefferson St, Sw Roanoke, VA 24016	PD	LAMPROS, MD, Jim Nicholas 402 United Va Bank Bldg Roanoke VA 24011	D	NOTTINGHAM III, MD, Clifford 2900 Peters Creek Road Roanoke, VA 24019	FP	SCHERTZ, MD, Gerald Lee 2013 S. Jefferson St, Sw Roanoke VA 24014	ON
GALE, MD, James Coter Roanoke Memorial Hosp Roanoke VA 24033	PTH	HILL, MD, Stephen L 1125 S. Jefferson St Roanoke, VA 24016	GS	LANIER, MD, Andrew Stephens Rd 3, Box 78B Floyd, VA 24091	EM	OAST, MD, Fred F 1917 Greenwood Rd Roanoke VA 24015	PUD	SCHLEUPNER, MD, Charles J. 3755 Kentland Dr, Sw Roanoke VA 24018	IM
GANDEE, MD, R. Wayne 2037 Crystal Spring Ave, Sw Roanoke, VA 24014	R	HOBACK, MD, Daniel Pflum 7533 Williamson Road, N W Roanoke, VA 24019	GP	LE PETER, MD, Allen Jos 1138 Second St Sw Roanoke VA 24016	OPH	OBSCHANSKY, MD, Margaret B 4450 Northridge St Ne Roanoke VA 24012	PH	SCRUGGS, MD, Hugh J Cancer Center Sw Va Roanoke VA 24033	TR
GARDNER, MD, John E 203 Med Arts Bldg Roanoke VA 24011	IM	HOLLINGSWORTH, MD, John H 1615 Franklin Road, Sw Roanoke VA 24016	CD	LESKO, MD, Edmund Michael Route 1, Box 300 Rocky Mount, VA 24151	AN	OWENS, MD, Richard S 803 Medical Arts Bldg Roanoke VA 24011	OPH	SEIF III, MD, Rahmet 707 Building 203 Roanoke VA 24011	GS
GARNER, MD, David S 2205 Brambleton Ave Sw Roanoke VA 24015	P	HULL, MD, George H P O Box 8119 Roanoke VA 24014	P	LEVY, MD, Myron S. 1802 Braeburn Drive Salem, VA 24153	IM	PAINE JR, MD, Robt Edward 808 Cherrywood Rd Salem VA 24153	IM	SHAFFER JR, MD, Lee W 1240 3rd St S W Roanoke VA 24016	ORS
GEIB, MD, Wayne A 20 Jingle Shell Lane Hilton Head Island SC 29928	PTH	HUMPHRIES III, MD, Marion K P O Box 1789 Roanoke VA 24008	OPH	LITWILLER, MD, Roger Wayne 5028 Falconridge Road Roanoke VA 24014	AN	PARK, MD, Jong Hee 3337 Clare Rd Sw Roanoke VA 24018	AN	SHAH, MD, Narendre Champakial V A Hospital Salem VA 24153	P
GILLILAND, MD, Charles D 1122 Second Street, Sw Roanoke, VA 24016	IM	HUMPHRIES JR, MD, William H 3200 Evergreen Lane, Sw Roanoke, VA 24018	EM	LOFTIN III, MD, Chas Ivey 1310 Third St S W Roanoke VA 24016	IM	PASLEY, MD, Faith R 2421 Crystal Spring Ave Roanoke, VA 24014	FP	SHAPIRO, MD, Andrew Dishert 1201 3rd St Sw Roanoke VA 24016	PD
GLASGOW, MD, Jean M Martin 510 Cherry St, #305 Bluefield, WV 24701	GP	HUMPHRIES, MD, Thos J 1802 Braeburn Dr Salem VA 24153	PD	LOUGHEED, MD, Marvin N Post Office 8567 Roanoke VA 24014	TR	PASLEY, MD, William W. 1234 Franklin Rd, Sw Roanoke, VA 24016	OBG	SHELTON, MD, John 2765 Bluefield Blvd, Sw Roanoke, VA 24015	EM
GLONTZ, MD, Gary Edwin 1603 Franklin Road, Sw Roanoke, VA 24016	IM	HURT JR, MD, John Omohundro 1906 Braeburn Drive Salem VA 24153	P	LOWE JR, MD, Richard H 105 Professional Bldg Roanoke VA 24014	U	PASSMORE, MD, Mildred R 4450 Northridge St Ne Roanoke VA 24012	PH	SHINER, MD, Philip Thompson 1615 Franklin Road, Sw Roanoke VA 24016	CD
GODWIN, MD, Gene Arthur 511 Blvd Salem VA 24153	GP	HURT, MD, Alvin Judson 117 Mcclanahan Street Roanoke VA 24014	OBG	LUCAS, MD, Davis C P O Box 8115 Roanoke VA 24014	A	PATTEN, MD, Robt Chester Route 1, Box 722 Troutville, VA 24175	FP	SHORTBRIDGE, MD, Chas M Box 1789 Roanoke VA 24008	OPH
GOOCH III, MD, Garrett G 1802 Braeburn Dr Salem VA 24153	OBG	HUTCHESON JR, MD, Robt S 1310 3D St S W Roanoke VA 24016	IM	LUEDKE, MD, George Wm 1112 Second Street, Sw Roanoke, VA 24016	P	PATTERSON JR, MD, Abram M. 2037 Crystal Spring Ave Roanoke, VA 24014	R	SIBLEY III, MD, Wm Lengley 1802 Braeburn Drive Salem VA 24153	GS
GORDGE, MD, Wm Noel 1201 3rd St S W Roanoke VA 24016	PD	HUTCHESON JR, MD, Jack Robert 2013 S Jefferson Street Roanoke VA 24014	IM	LUTHER, MD, Burton Lowe 3048 Brambleton Ave W Roanoke VA 24015	GP	PAUZE, MD, John Alexander 2519 Creston Ave Sw Roanoke VA 24015	FP	SIBLEY, MD, Wm Lengley 823A Duke Of Gloucester St Sw Roanoke VA 24014	GS
GRADY, MD, Roger Clifton 1315 Second St Sw Roanoke VA 24016	PD	IRVIN JR, MD, Robt Wheary Roanoke Memorial Hospital Roanoke VA 24009	OBG	MAGIER, MD, Nine G V A Hosp Salem VA 24153	P	PENDLETON, MD, John W. 2037 Crystal Spring Ave Roanoke, VA 24014	IM	SIEBER, MD, Homer Alden 1603 Franklin Rd Roanoke VA 24016	CD
GRAY, MD, William G 310 Washington Ave, Sw Roanoke, VA 24016	CHP	IVEY JR, MD, Henry Reese 204 S Maple Street Vinton, VA 24179	FP	MANLEY JR, MD, Walter F 30 Franklin Road, Sw Roanoke VA 24011	D	PERKINS, MD, Marvin E P O Box 126 Fincastle VA 24090	P	SILBER, MD, Gershon P O Box 3036 Roanoke, VA 24015	P
GRAYSON JR, MD, Richard Jos P O Box 8306 Roanoke VA 24014	OTO	JACOBSON, MD, Abraham M. 17 Highland Avenue, S W Roanoke, VA 24016	IM	MANLEY, MD, Walter F 2902 Crystal Spring Ave Roanoke VA 24014	D	PETERSON JR, MD, Chas Hanson 2129 Rosalind Ave Sw Roanoke VA 24014	P O	SINGER, MD, Lewis Jay 1138 2nd Street, Sw Roanoke, VA 24016	PD
GRAYSON, MD, Wayne Edward 3142 Brambleton Ave Roanoke, VA 24018	FP	JENNINGS JR, MD, C Leon 4231 Colonial Ave, Sw Roanoke, VA 24018	OBG	MARTIN, MD, John Albert 2037 Crystal Sp Ave Sw Roanoke VA 24014	R	PIERCE, MD, Douglas Edward 1201 3rd St Sw Roanoke VA 24016	PD	SISK, MD, Michael Anthony 2601 Franklin Rd Sw Roanoke VA 24014	CHN
GREENSTEIN, MD, Raphael H 5139 Cherokee Hills Dr Salem VA 24153	IM	JOHNSON, MD, Earl Robt 2006 Windsor Ave, Sw Roanoke VA 24015	IM	MAYSON JR, MD, Preston B 4906 Buckhorn Drive Roanoke VA 24014	R	POOLEY, MD, Robt Earl Box 13367 Roanoke VA 24033	PTH	SLEMP JR, MD, Andrew Alfred 1101 First St Sw Roanoke VA 24016	GS
GREER, MD, Wm Crockett 3112 Peters Creek Rd Nw Roanoke VA 24019	GP	JOHNSON, MD, Frank Mitchell Box 7236 Roanoke VA 24019	OS	MC CAUSLAND, MD, Alexander 609 S Jefferson St Roanoke VA 24011	A	PRUNER, MD, Robert A 1240 Third St Roanoke VA 24016	ORS	SNEAD, MD, James Given 3232 Fordham Rd Sw Roanoke VA 24014	R
GUELZOW, MD, Kurt W L 3320 Franklin Road, Sw Roanoke VA 24014	OPH	JOHNSON, MD, Harry I 1315 2D St S W Roanoke VA 24016	CD	MC DANNALD JR, MD, Eugene R 1125 S. Jefferson Street Roanoke VA 24016	GS	QUIOCO, MD, Heathcliff M. 312 North Main Street Rocky Mount, VA 24151	GS	SPETZLER, MD, Bertram 1802 Breburn Drive Salem, VA 24153	ORS
GUILFOYLE, MD, Francis M 1201 3rd St S W Roanoke VA 24016	PD	JOHNSON, MD, Walter Smith 127 Mc Clanahan St S W Roanoke VA 24014	GS	MELCHIONNA, MD, Olin Richard 3022 Pioneer Rd N W Roanoke VA 24012	GP	RACE, MD, Donald John 1138 2nd St Sw Roanoke VA 24016	OPH	STARR, MD, John Walter 1615 Franklin Road, Sw Roanoke VA 24016	CD
GUNZENHAUSER, MD, Leslie 1111 South Jefferson St Roanoke VA 24016	IM	JONES, MD, Benj Newman 1802 Braeburn Dr Salem VA 24153	EM	MEYER, MD, Julien H 2118 Rosalind Ave., S. W. Roanoke VA 24014	OBG	RAGIONE, MD, Jos Alfred Box 237-A Moneta, VA 24121	OM	STAVOLA, MD, Anthony R 1836 Greenwood Road Sw Roanoke VA 24015	FP
HAGAN, MD, Margaret Lee P O Box 280 Buchanan, VA 24066	FP	JONES, MD, Daniel Relph 204 South Maple St Vinton VA 24179	FP	MILLER, MD, John Milton 1802 Braeburn Dr Salem VA 24153	IM	REMANABAN, MD, Teodulo 3839 Bosworth Drive, Sw Roanoke, VA 24014	AN	STEPHENS, MD, Wesley G Box 157 Troutville VA 24175	EM
HAGUE, MD, Frank Jos 3044 Carolina Ave Sw Roanoke VA 24014	AN	JOSEFIK, MD, Eugene Jos 4962 Foxridge Rd Sw Roanoke VA 24014	PTH	MINICHAN, MD, David Parrish 1234 Franklin Rd S W Roanoke VA 24016	GS	RENICK, MD, Ole Wibholm Box 1789 Roanoke, VA 24008	OTO	STEVENS JR, MD, Ward Wm 2601 Franklin Rd Sw Roanoke VA 24014	NS
HAGY, MD, John Albert Rt 4 Rocky Mount VA 24151	FP	KAGEY, MD, William Joseph 1803 Braeburn Drive, Sw Salem, VA 24153	PD	MIRENDA JR, MD, William M 1240 Third Street, Sw Roanoke, VA 24016	ORS	RICE, MD, James Davies 2855 South Jefferson St, Sw Roanoke VA 24014	R	STEWART, MD, Bruce Neal 1111 South Jefferson St Roanoke VA 24016	PUD

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<b>STONE JR</b> , MD, Harry Benj 2215 Brambleton Ave Sw Roanoke VA 24015	OPH	<b>WILEY</b> , MD, Roger P. 3557 Peakwood Dr., Sw Roanoke, VA 24014	DR	<b>ANDES</b> , MD, Geo Calvert 252 E Grattan St Harrisonburg VA 22801	OPH	<b>HOTCHKISS</b> , MD, Wm J Box 397 Broadway VA 22815	GP	<b>SEWICK</b> , MD, Richard E.N. 1041 South Main Street Harrisonburg, VA 22801	OBG
<b>STONE</b> , MD, Wm Conrad 4019 Lake Drive Roanoke VA 24018	OPH	<b>WILKS</b> , MD, John Wm 1234 Franklin Rd S W Roanoke VA 24016	GYN	<b>ARMENTROUT</b> , MD, Clement S 468 Ott St Harrisonburg VA 22801	GYN	<b>HUFFMAN</b> , MD, Rutus Clyde P O Box 237 Bridgewater VA 22812	FP	<b>SENFIELD</b> , MD, Richard Maxon 1840 East Market Street Harrisonburg, VA 22801	AN
<b>STRICKLER</b> , MD, Frank Andes 165 Lee Ave, Ne Roanoke VA 24012	P	<b>WILLIAMS II</b> , MD, Edwin Leon 4536 Greenlee Rd Sw Roanoke VA 24018	GS	<b>BLAY</b> , MD, Andrew Brian 108 Flint Avenue Harrisonburg, VA 22801	EM	<b>KENNEL</b> , MD, Elmer Elwood 1031 South Main St Harrisonburg VA 22801	GS	<b>SHANK</b> , MD, David Lee 1186 Portland Dr. Harrisonburg, VA 22801	EM
<b>STRONG</b> , MD, Thomas E 1240 3rd St S W Roanoke VA 24016	ORS	<b>WILLIAMS II</b> , MD, Samuel 213 Mcclanahan St, Sw, #404 Roanoke, VA 24014	GS	<b>BOOTH</b> , MD, Hobson G 1041 South Main St Harrisonburg, VA 22801	OBG	<b>KENSELL</b> , MD, Ralph F. 110 Hemlock Harrisonburg, VA 22801	FP	<b>SHEAP</b> , MD, Christopher Newkirk 1015 Harrison St Harrisonburg VA 22801	D
<b>SUDRANSKI</b> , MD, Herbert F 5151 Carriage Dr Sw Roanoke VA 24016	OPH	<b>WILLIAMS</b> , MD, Donald Richard 511 Boulevard Salem VA 24153	FP	<b>BROOKS</b> , MD, Charles Harris 847 Cantrell Avenue Harrisonburg, VA 22801	IM	<b>KIDWELL</b> , MD, John Aaron Rt 1 Box 178B Port Republic VA 24471	EM	<b>SHOWALTER</b> , MD, Carl Robt 1031 S Main St Harrisonburg VA 22801	P
<b>SYDNOR</b> , MD, J Brantley P O Box 8306 Roanoke VA 24014	OTO	<b>WINBORNE JR</b> , MD, Roger M 374 Woods Ave Sw Roanoke VA 24016	IM	<b>BRUNK</b> , MD, James Robt 1031 S Main St Harrisonburg VA 22801	PUD	<b>KLIM</b> , DO, Philip A 402 Garber's Church Road Harrisonburg, VA 22801	OST	<b>SHOWALTER</b> , MD, Samuel G Family Health Center Weyers Cave VA 24486	FP
<b>TAYLOR</b> , MD, Roger Scott 2308 Crowncrest Drive Richmond, VA 23233	EM	<b>WINN JR</b> , MD, Thomas M. 4231 Colonial Ave, Sw Roanoke, VA 24018	OBG	<b>BRYANT</b> , MD, Robt Singleton 725 S Mason St Harrisonburg VA 22801	IM	<b>KNISS</b> , MD, Mark Allan 1000 Chicago Avenue Harrisonburg VA 22801	FP	<b>SMITH JR</b> , MD, Richard H. Route 2, Box 295 N Bridgewater, VA 22812	EM
<b>TAYLOR</b> , MD, Smith Davis Rt 1 Box 407 Fincastle VA 24090	FP	<b>WISMAN</b> , MD, Wm Robt 1506 Franklin Rd S W Roanoke VA 24016	OBG	<b>BURT</b> , MD, Leslie Stephen 1031 S Main St, #29 Harrisonburg VA 22801	PD	<b>KOHRING</b> , MD, Regis Clarke 1840 East Market St Harrisonburg VA 22801	AN	<b>SMITH</b> , MD, Joseph Douglas Route 2, Box 32K Bridgewater, VA 22812	FP
<b>TEAGUE</b> , MD, Nelson Stone 1234 Franklin Rd Roanoke VA 24016	U	<b>WITTEN</b> , MD, James A 3802 Bosworth Dr Roanoke VA 24014	PUD	<b>BYERS</b> , MD, Francis L 1049 S Main St Harrisonburg VA 22801	GP	<b>LAMBERT</b> , MD, Lynn D. 2400 Port Road Harrisonburg, VA 22801	PD	<b>SMITH</b> , MD, Michael E. 85 Rex Road Harrisonburg VA 22801	DR
<b>THOMAS</b> , MD, Bruce Richard 2037 Crystal Spring Ave Roanoke VA 24014	R	<b>WOOD</b> , MD, John Robert 707 S Jefferson St, Sw Roanoke, VA 24008	OPH	<b>CALDWELL</b> , MD, Paul Chas 417 Paul St Harrisonburg VA 22801	DR	<b>LAMONT</b> , MD, Jeffrey H. 1031 South Main Street Harrisonburg, VA 22801	PD	<b>STAUFFER</b> , MD, John M P O Box 388 Timberville, VA 22853	FP
<b>THOMAS</b> , MD, Wm Odell 2037 Crystal Sp Ave Roanoke VA 24014	DR	<b>YATES JR</b> , MD, Harry Robt 2037 Crystal Spring Roanoke VA 24014	IM	<b>CALE</b> , MD, William F 847 Cantrell Avenue Harrisonburg VA 22801	IM	<b>LAREAU</b> , MD, Eugene Raymond 1031 South Main St St 11 Harrisonburg VA 22801	GS	<b>STORY</b> , MD, Wm Henry 1840 East Market St Harrisonburg VA 22801	AN
<b>TICE</b> , MD, Wm Preston 1305 Lakewood Drive Roanoke VA 24015	NS	<b>YOUNG JR</b> , MD, Charles A 122 Mountain Ave S W Roanoke VA 24016	OPH	<b>CALLAHAN</b> , MD, Donald Morris 1031 S Main St Ste 10 Harrisonburg VA 22801	R	<b>MAJLESSI</b> , MD, Heshmatolah 170 South Main Street Harrisonburg, VA 22801	N	<b>TALBOT</b> , MD, Wm Hanna 235 Cantrell Avenue Harrisonburg VA 22801	PTH
<b>TORRE</b> , MD, Anthony Vincent Lewis Gate Hse-1900 Electric Salem VA 24153	PTH	<b>ROCKBRIDGE</b>		<b>CANTER JR</b> , MD, Noland M 456 Ott Street Harrisonburg VA 22801	R	<b>MALONE</b> , MD, Jonathan K. 861 Cantrell Ave, #A Harrisonburg, VA 22801	ORS	<b>THOMPSON</b> , MD, James Walker 114 South Main St Bridgewater VA 22812	IM
<b>TROSTLE</b> , MD, Thomas F 5013 Falcon Ridge Road Roanoke VA 24014	AN	<b>BRUSH JR</b> , MD, Edward V 2 E Washington St Lexington VA 24450	GP	<b>CHALAM</b> , MD, Ramesh 1840 East Market St Harrisonburg VA 22801	AN	<b>MANSFIELD</b> , MD, John Bristow 725 South Mason Street Harrisonburg, VA 22801	GS	<b>TORKELSON</b> , MD, Leit Oscar Route 1, Box 240 D Mount Crawford, VA 22841	CD
<b>TUCK</b> , MD, Kenneth Douglas 3320 Franklin Road, Sw Roanoke VA 24014	OPH	<b>COLEMAN</b> , MD, Howe Reese P O Box 908 Lexington VA 24450	OPH	<b>CHAPPELL JR</b> , MD, George E 311 Paul Street Harrisonburg VA 22801	ORS	<b>MC DONALD</b> , MD, Robert M. 475 Myers Avenue Harrisonburg VA 22801	PD	<b>VEST</b> , MD, Timothy Keith 847 Cantrell Avenue Harrisonburg, VA 22801	GE
<b>VANCE</b> , MD, Saml Franklin 1640 Persinger Road Roanoke VA 24015	HEM	<b>COUPER</b> , MD, John Lee 327 Overhill Ave Lexington VA 24450	AN	<b>COMER</b> , MD, Francis E. Box 40, Route 7 Harrisonburg VA 22801	PD	<b>MILLER</b> , MD, Chas S Box 165 Elkton VA 22827	GP	<b>WAMPLER</b> , MD, Garland Jos Box 2500 Staunton, VA 24401	GP
<b>VARNER</b> , MD, John D 213 Mcclanahan St Roanoke VA 24014	NS	<b>DICK</b> , MD, W Barton 110 Houston Street Lexington VA 24450	ORS	<b>CRAUN JR</b> , MD, Galen G. 1015 Harrison Street Harrisonburg VA 22801	ORS	<b>MORESHEAD</b> , MD, Jon Ashton 1000 Chicago Avenue Harrisonburg VA 22801	FP	<b>WEIDIG JR</b> , MD, George Louis 1015 Harrison Street Harrisonburg VA 22801	FP
<b>VERMILLION</b> , MD, Robert L. 2118 Rosalind Ave, Sw Roanoke, VA 24014	OBG	<b>ECHOLS</b> , MD, Wm Beecher 110 Houston St, #A Lexington VA 24450	OTO	<b>CRAUN</b> , MD, Galen Glick Rt 1 Box 92 Harrisonburg VA 22801	GS	<b>MORRISON</b> , MD, A. Glenn 235 Cantrell Ave. Harrisonburg, VA 22801	PTH	<b>WHITE</b> , MD, Gordon Osler 170 S Main St Harrisonburg VA 22801	N
<b>VICTOR</b> , MD, Fen'n Ernest 516 11th St N W Roanoke VA 24017	GP	<b>FEDEMAN</b> , MD, Frederick A 2 E Washington St Lexington VA 24450	GP	<b>DEYERLE</b> , MD, Henry Price 312 S Main St Harrisonburg VA 22801	GS	<b>NASH</b> , MD, Beverly W Timberville VA 22853	FP	<b>WHITEHEAD JR</b> , MD, David C. 1015 Harrison St Harrisonburg VA 22801	FP
<b>WADE</b> , MD, Evelyn Henry Clark 3568 Peakwood Dr Sw Roanoke VA 24014	P	<b>FOX</b> , MD, Kurt Johannes Medical Bldg Fairfield VA 24435	GP	<b>DUMLER JR</b> , MD, John Chas 1015 Harrison Street Harrisonburg VA 22801	D	<b>NIPE</b> , MD, Geo Maynard 1031 S Main St Harrisonburg VA 22801	OBG	<b>WINE</b> , MD, Jean Fennell 57 Paul Street Harrisonburg VA 22801	IM
<b>WADE</b> , MD, Frank Alton 1802 Braeburn Dr Salem VA 24153	IM	<b>HARRALSON</b> , MD, John David 110 Houston Street Lexington VA 24450	OBG	<b>EAGLE</b> , MD, John Russell 640 South Main St Harrisonburg VA 22801	P	<b>PEREZ</b> , MD, Jose R. 861 Cantrell Ave Harrisonburg VA 22801	U	<b>WITMER</b> , MD, Daniel G. 1041 S. Main St Harrisonburg VA 22801	OBG
<b>WAKAT</b> , MD, Marshall A. 2037 Crystal Spring Ave Roanoke VA 24014	NM	<b>HEDRICK</b> , MD, Thos B 2141 Sycamore Avenue Buena Vista VA 24416	GP	<b>EGGLESTON</b> , MD, Robert B. 245 Newman Ave Harrisonburg VA 22801	OPH	<b>PERRY</b> , MD, Danny L P O Box 388 Timberville, VA 22853	FP	<b>YODER JR</b> , MD, Paul Roy 1031 South Main St Harrisonburg VA 22801	OPH
<b>WALKE</b> , MD, John Tabb 1802 Braeburn Dr Salem VA 24153	PD	<b>IRONS JR</b> , MD, Robert Price Rockbridge Professional Bldg Lexington, VA 24450	GS	<b>ESHLEMAN</b> , MD, Merle W 1501 Va Ave, Apt 249 Harrisonburg VA 22801	GP	<b>PRESTON</b> , MD, Robert Willard 1031 S Main Harrisonburg VA 22801	OPH	<b>YODER</b> , MD, Gene Lee 1031 S. Main Street Suite 28 Harrisonburg, VA 22801	IM
<b>WALKER</b> , MD, Rome Haward 3259 White Oak Rd Roanoke, VA 24014	GS	<b>IRONS</b> , MD, Robt P Rockbridge Prot Bldg Lexington VA 24450	GS	<b>EVANS</b> , MD, James Dewitt 1031 South Main Street Harrisonburg VA 22801	FP	<b>QUISLING</b> , MD, Richard Warren 395 Wallace Road Nashville, TN 37211	OTO	<b>YODER</b> , MD, Paul T. 1000 Chicago Avenue Harrisonburg VA 22801	GPM
<b>WALL</b> , MD, Geo Hampton 1802 Braeburn Dr Salem VA 24153	GE	<b>KENNAN</b> , MD, Thos F Star Route B, Box 44 Staunton, VA 24401	GP	<b>FLETCHER</b> , MD, Wm Paul 1429 S Main St Harrisonburg VA 22801	FP	<b>RAPP</b> , MD, Raymond Edward 1840 East Market St Harrisonburg VA 22801	AN	<b>ZAPANTA</b> , MD, Conrado R 831 Cantrell Avenue Harrisonburg VA 22801	OTO
<b>WALLENBORN JR</b> , MD, Peter A P O Box 8306 Roanoke VA 24014	OTO	<b>LARSEN</b> , MD, Geo Douglas 2 Courtland Center Lexington VA 24450	OBG	<b>FOX</b> , MD, Frederick Louvane 1041 South Main Street Harrisonburg VA 22801	ORS	<b>READ</b> , MD, Marc Edward Shenandoah Co Mem Hosp Woodstock, VA 22664	R	<b>ZIRKLE JR</b> , MD, Walter M. 1041 S. Main St Harrisonburg VA 22801	OBG
<b>WALTON</b> , MD, David Clark 5020 Grandin Road Roanoke VA 24018	PDA	<b>LEWIS</b> , MD, Thomas Cary Rockbridge Prot Bldg Lexington, VA 24450	IM	<b>GARDNER</b> , MD, Jos Erskin 1031 S Main Harrisonburg VA 22801	FP	<b>REILLY</b> , MD, Michael John 1031 South Main St, #9 Harrisonburg VA 22801	OBG	<b>SCOTT</b>	
<b>WARD</b> , MD, Wm Caldwell 3142 Brambleton Ave Sw Roanoke VA 24018	FP	<b>MC CLUNG JR</b> , MD, Oscar H Rockbridge Prot Bldg Lexington VA 24450	GP	<b>GEARING JR</b> , MD, Frank W 813 Oak Hill Dr Harrisonburg VA 22801	R	<b>REISH</b> , MD, Wm Edwin 4060 South Main St Harrisonburg VA 22801	GP	<b>ADKINS</b> , MD, Bruce Richard 218 D U.S. Hwy 23 North Weber City, VA 24251	GP
<b>WATTS</b> , MD, Earl Wilson 1802 Braeburn Drive Salem VA 24153	OM	<b>MC CLUNG</b> , MD, John Houston Box 65 Glasgow VA 24555	GP	<b>GLICK JR</b> , MD, John T Box 397 Broadway VA 22815	GP	<b>RIDDEL JR</b> , MD, Clifford T 401 West Bank St Bridgewater VA 22812	EM	<b>GRIGSBY</b> , MD, Wm Paul 1735 Fort Henry Drive Kingsport TN 37660	GS
<b>WEAVER</b> , MD, Edgar Newman 1315 Second Street, Sw Roanoke VA 24016	NS	<b>MICOU</b> , MD, Lewis A 3100 Shore Drive, Apt 1017 Virginia Beach, VA 23451	GP	<b>GRAVES V</b> , MD, Asa Wesley Lacey Spring VA 22833	GP	<b>SANTOS</b> , MD, Dominador S. 1356 1/2 South Main St Harrisonburg VA 22801	GS	<b>HAMPTON</b> , MD, Hobert M. 150 Locust Weber City, VA 24251	GP
<b>WELLER</b> , MD, Wm Franklin Box 8065 Roanoke VA 24014	DR	<b>OLD III</b> , MD, Wm Whitehurst Rockbridge Prot Bldg Lexington VA 24450	GS	<b>HARPER II</b> , MD, G. William 1015 Harrison Street Harrisonburg VA 22801	FP	<b>SCHLABACH</b> , MD, Walter E 1139 Waterman Drive Harrisonburg VA 22801	GS	<b>HONEYCUTT JR</b> , MD, Grover C P O Box 278 Gate City VA 24251	PH
<b>WELLS</b> , MD, Hugh Haynsworth P O Box 13367 Roanoke, VA 24033	PD	<b>PICKRAL</b> , MD, Robert M. 205 Johnstone Street Lexington, VA 24450	FP	<b>HARPER</b> , MD, Eugene Jutson 1031 S Main Street Harrisonburg VA 22801	GS	<b>SCHULTZ</b> , MD, Joyce A. 1031 S. Main Street Suite 6 Harrisonburg, VA 22801	PS	<b>SOUTHSIDE VIRGINIA</b>	
<b>WHELESS</b> , MD, James Elijah 1603 Franklin Rd S W Roanoke VA 24016	IM	<b>RAST</b> , MD, Philip R. Lex Buena Vista Shopping Park Lexington, VA 24450	U	<b>HEARN</b> , MD, John T Montevideo Clinic Penn Laird VA 22846	IM	<b>SCHULTZ</b> , MD, Robt Gwynn Med Arts Bldg Harrisonburg VA 22801	OBG	<b>ADAMS</b> , MD, Allan B P O Box 242 Gulf Shores, AL 36542	R
<b>WHITE</b> , MD, Paul Fletcher 609 S. Jefferson St Roanoke VA 24011	A	<b>SLAUGHTER</b> , MD, Arthur Robert 2252 Magnolia Avenue Buena Vista, VA 24416	FP	<b>HELBERT</b> , MD, Hollen G 1031 S Main St Harrisonburg VA 22801	R	<b>SEASE JR</b> , MD, Cyril Iredel 725 S Mason St Harrisonburg VA 22801	U	<b>ANDREW</b> , MD, Theodore C P O Box 815 Hopewell VA 23860	GP
<b>WHITMAN JR</b> , MD, Wm Rush Route 1, Box 218 Goodview, VA 24095	GS	<b>WEDDLE</b> , MD, William E 2252 Magnolia Avenue Buena Vista, VA 24416	FP	<b>HENDERSON</b> , MD, Chas Henry Medical Arts Bldg Ste 10 Harrisonburg VA 22801	ORS	<b>SEASE</b> , MD, James Richard 725 S Mason St Harrisonburg VA 22801	GS	<b>ANTONIA</b> , MD, Eugenio V. 4075 Ruffin Road Hopewell, VA 23860	GS

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ASHBY, MD, Chas Chandler P O Box 339 Dimwiddle VA 23841	GP	DREWRY, MD, David B 111 Morton St Petersburg VA 23805	PD	HO, MD, Thuy Nguyen 603 S. Sycamore St Petersburg, VA 23803	OBG	MOORE, MD, Earle Winston P O Box 577 Chase City VA 23924	FP	SHIEH, MD, Frank F 603 Sycamore St Petersburg VA 23803	OBG
BADIN III, MD, Nicholas 1840 Westchester Dr Petersburg VA 23803	R	DURRANI JR, MD, Waheed 709 Cedar Lane Rt 1 Hopewell, VA 23860	P	HOLDEN, MD, Bobby G 520 South Sycamore St Petersburg VA 23803	GP	MOORE, MD, Kermil J P O Box 2088 Petersburg VA 23803	GP	SHIM, MD, Jaimoon M. 406 North 6th Ave Hopewell, VA 23860	OTO
BANE, MD, Earle M 319 West Church St Lawrenceville VA 23868	GP	EAPEN, MD, George 401 West Ave Emporia, VA 23847	CD	HOOVER, MD, Herbert C 1833 S Sycamore St Petersburg VA 23805	AN	MOORE, MD, Ray A 121 E 3D St Farmville VA 23901	IM	SHIM, MD, Young S. 602 N 6th Avenue Hopewell, VA 23860	IM
BARNEWELL, MD, Benj Burdee 40 Liberty St Petersburg VA 23803	GS	EAPEN, MD, Sari 103 Shore Dr Emporia, VA 23847	PM	HOWELL, MD, Taimedje Rudolph 3325 Osborn Road Chester, VA 23831	R	MORGAN III, MD, Walter Edward 408 S Sycamore St Petersburg VA 23803	OPH	SIRIWATHARANGON, MD, C 400 Woodland Road Hopewell, VA 23860	OBG
BAUGH JR, MD, Emerson D. P O Box 310 Kenbridge VA 23944	FP	EASTERLING, MD, John G 207 E Cawson St Hopewell VA 23860	GS	HU, MD, Anthony Wen-Shih John Randolph Hospital Hopewell, VA 23860	PTH	MOSELEY, MD, Chas Hilary 530 S Sycamore St Petersburg VA 23803	OBG	SKAGGS, MD, Jerome D 301 W Broadway Hopewell VA 23860	FP
BAYNARD, MD, Melvin G Truck St Lawrenceville VA 23868	ABS	EHRENWORTH, MD, Adolphe M 1964 Wakefield St Petersburg VA 23805	R	ISHIZAWAR, MD, Yorkcay C. 510 S Sycamore, #B Petersburg, VA 23803	GS	MUNOZ, MD, Anthony Jos 420 E Third St Farmville VA 23901	GS	SLOAN, MD, Wm Stringfield 416 Petersburg Mutual Bldg Petersburg VA 23803	IM
BERGEN, MD, Frederick 116 S Sycamore St Petersburg VA 23803	TS	EMILIANI, MD, N.A. 312 S. Sycamore Street Petersburg, VA 23803	P	JACOBS, MD, Erwin Melvin 510 South Sycamore St Petersburg VA 23803	N	MURTHY, MD, G.S. 116 South Sycamore St Petersburg, VA 23803	PD	SMITH, MD, Nelson Montgomery 116 S Sycamore St Petersburg VA 23803	GS
BIGLEY JR, MD, H Alan 700 S.Sycamore St Petersburg VA 23803	U	ENDE, MD, Milton Facp 121 S Market St Petersburg VA 23803	IM	JENSEN, MD, Edward Walter 4810 Bruce Road Chester, VA 23831	IM	NASE, MD, Harold Wallace Box 504 Farmville, VA 23901	GS	SMITH, MD, Robt Sullins Route 1 Box 16 Dimwiddle VA 23841	FP
BISHOP, MD, Wm Branch 319 W. Church St Lawrenceville VA 23868	GP	FARBER, MD, Herman Wm 111 Morton St Petersburg VA 23803	PD	JOHNSON, MD, Alfred G 444 Halifax St Petersburg VA 23803	GS	NEIFELD, MD, David M. P O Box 1079 Hopewell, VA 23860	FP	SQUIRE, MD, Peter W 219 Weaver Ave Emporia VA 23847	FP
BLAKE, MD, Michael Clarence 415 West Broadway Hopewell, VA 23860	GS	FIELD, MD, Bolling Jones 507 S Sycamore St Petersburg VA 23803	CD	JOHNSON, MD, Allen C. 1600 Walton Road Petersburg, VA 23805	DR	O BRIEN, MD, Clyde G Appomattox VA 24522	GP	STINNETT, MD, Rodney G. Norton Community Hosp Norton, VA 24273	R
BOAZ JR., MD, Beverly Gilly 1834 Fort Rice St Petersburg VA 23803	AN	FEMINELLA JR., MD, John Geo 700 S.Sycamore St Petersburg VA 23803	U	JONES, MD, J. Kipling 3224 Hastings Road Petersburg VA 23805	CHP	O'DONNELL, MD, Philip 510 S Sycamore St Petersburg, VA 23803	N	STURMER, MD, Frederick Chas 416 Durant Street South Hill VA 23970	GS
BOUTROS, MD, Samir B.G P O Box 25 South Hill, VA 23970	U	FLAHERTY, MD, Michael J. 712 West Broadway Hopewell, VA 23860	HEM	JUDY, MD, Sami Benj Box 337 Clerksville VA 23927	FP	PEARCE JR., MD, Carney C 1661 Blair Road Petersburg, VA 23803	OBG	SUMA, MD, Ben P. P O Box 1832 Petersburg VA 23803	OTO
BOWLES, MD, James William 408 S. Sycamore Street Petersburg, VA 23803	OPH	GEAR, MD, Arthur Sewell Post Office Box 536 South Hill, VA 23970	CD	KAWAKAMI, MD, Fumikazu 1140 Varnum St, Ne Washington, DC 20017	IM	PERINI, MD, Clinton J 700 S Sycamore St, #6 Petersburg, VA 23803	R	SUPETRAN, MD, Virgilio Conlu 502 N 6th Ave Hopewell VA 23860	OBG
BRAND, MD, Rolf 212 Mecklenburg Avenue South Hill, VA 23970	GP	GOLDBERG, MD, Jay Stephen 3731 B Blvd Colonial Heights VA 23834	IM	KEELING, MD, Robt D P O Box 180 South Hill VA 23970	GP	PEVSNER, MD, Paul Hershel P O Box 604 Farmville, VA 23901	OTO	SUSLICK, MD, Randall Hugh P O Box 577 Chase City, VA 23924	GP
BRANTLEY, MD, Aurelius Walter 205 Cypress Ave Franklin VA 23851	FP	GOMEZ, MD, Maximmo 1839 Sherwood Road Petersburg VA 23805	PD	KIRKLAND JR, MD, James A 6 Doctors Dr Emporia VA 23847	GP	PHIPPS, MD, Glenn Ward 408 S Sycamore St Petersburg VA 23803	R	TALEGAONKAR, MD, S K 304 West Broadway Hopewell VA 23860	OPH
BRAXTON, MD, Herman H P O Box 278 Chase City VA 23924	FP	GONZALES, MD, Manuel B 801 South Adams Street Petersburg VA 23803	PTH	KRAMER, MD, Marc Stephen 3 Tuckahoe Blvd Richmond, VA 23226	FP	POWELL JR., MD, Clarence L Southampton Med Bldg Franklin VA 23851	OPH	TANNER JR., MD, Henry M P O Box 624 South Hill VA 23970	US
BRICKHOUSE, MD, Albert T 207 E Cawson St Hopewell VA 23860	GP	GONZALES, MD, Patricie D 4202 Quebec Ave Prince George Ve 23875	PD	KRISHNAMURTHY, MD, Kelale S 29 Morton Avenue Petersburg VA 23805	IM	PRINCE, MD, John Stuart Po Box 508 Emporia VA 23847	US	TAWFICK, MD, Mohammed A 101 Morton Ave Petersburg VA 23803	ORS
BRIDGFORTH, MD, Lewis Wm Box A-F Victoria VA 23974	GP	GONZALEZ, MD, Juan M 611 Third Ave Farmville VA 23901	GP	LA ROCHE II, MD, Ripon W. P O Box 506 Farmville, VA 23901	OPH	QUICK, MD, Cedric Albert 700 S Sycamore St, #11 Petersburg VA 23803	GP	TAYLOR, MD, Francis N 603 Virginia First Building Petersburg VA 23803	GP
BURWELL, MD, Bronwen 1570 Brandon Ave Petersburg, VA 23805	PD	GORDON, MD, Edward T P O Box 603 Farmville VA 23901	PD	LACY II, MD, Matthew L Box 95 South Hill VA 23970	GS	QUITOQUIT, MD, Eftren Aninag Emporia Medical Associates Emporia VA 23847	OTO	THUNG, MD, Nalda Sylvia 1632 Wilton Road Petersburg VA 23803	GS
CARTWRIGHT, MD, Crosby W 733 Halifax Street Emporia VA 23847	GP	GRAHAM JR., MD, Sami Alan P O Box 347 Farmville VA 23901	PH	LAYMAN, MD, David Arthur 301 W Broadway Hopewell VA 23860	FP	RAMIREZ, MD, Michael E. Powhatan, VA 23139	FP	TOWNES, MD, Chas Henry 20009 Oakland Ave Colonial Heights VA 23834	OS
CHAMBERLAIN, MD, Chas Wm Willmary Court Waverly VA 23890	EM	GRELLA JR, MD, Benjamin Route 2, Box 327 Bracey, VA 23919	DR	LEE, MD, Ming S 10904 Appletree Lane Hopewell VA 23860	GP	REARDON, MD, Patrick A. 111 Morton Avenue Petersburg, VA 23805	PD	TURNER III, MD, John Mills 203 Randolph Street Farmville VA 23901	OS
CHILDREY, MD, Stephen P O Box 68 Chase City VA 23924	GP	GRIFFIN JR., MD, Harvey Lee Petersburg Gen Hosp Petersburg VA 23803	PTH	LEE, MD, Young 603 S. Sycamore St Petersburg, VA 23803	GP	REDDY, MD, P. Vijay 510 S Sycamore St, #D Petersburg VA 23803	FP	VANICHKACHORN, MD, Sukri P O Box 489 South Hill VA 23970	OBG
CHIU, MD, Ming Sung 510 S. Sycamore St Petersburg VA 23803	IM	GRISWOLD, MD, Martha A. 700 S Sycamore, #3 Petersburg, VA 23803	IM	LEGROW, MD, Wynne V.E. 306 Weaver Ave Emporia, VA 23847	N	REDDY, MD, Pannala J M 510 South Sycamore Street Petersburg VA 23803	US	VAUGHAN, MD, Stephen F. 18 Shore Street Petersburg, VA 23803	ORS
CHOU, MD, Yi-Nan 11008 Whitepine Drive Hopewell VA 23860	CD	GRIZZARD, MD, Wm Sami 530 S Sycamore St Petersburg VA 23803	OBG	LEWIS, MD, Cyrus Patrick 201 Temple Ave Colonial Heights VA 23834	FP	RICE, MD, Benjamin Holt 700 S Sycamore St Petersburg VA 23803	GS	VICK JR., MD, Clyde Whitley 116 S Sycamore St Petersburg VA 23803	FP
COHEN, MD, Alvin 531 S Sycamore St Petersburg VA 23803	IM	GUILLERMIN, MD, John 1570 Brandon Avenue Petersburg VA 23805	NS	LIN, MD, Hsing-Wu 700 Okuma Drive Chester, VA 23831	CD	RICHARD, MD, Louis E 1964 Wakefield St Petersburg VA 23805	R	WADSWORTH, MD, James D P O Box 413 Colonial Heights, VA 23834	R
COLLMANN, MD, Warren X 108 4th St Farmville VA 23901	GS	GULMATICO, MD, Oscar B 913 B W Danville St South Hill VA 23970	PD	LINK, MD, Garnett Wm 114 Lakeshore Dr Petersburg VA 23803	CD	ROBBINS, MD, Clayton Asa 1231 Rome Street Petersburg, VA 23803	CD	WALKER, MD, Thos Andrew 605 Lakeside Dr Emporia VA 23847	GP
CONCODOIRA, MD, Joseph A. 615 West Broadway Hopewell, VA 23860	U	HAINES, MD, David Morrill P O Box 1835 Petersburg VA 23803	ORS	LUM, MD, Natalie Inge P O Box 1748 Petersburg VA 23805	PD	ROEBUCK, MD, Jerome Barland 408 S Sycamore Petersburg VA 23803	OPH	WARREN, MD, Rufus Hawkins 520 S Sycamore St Petersburg VA 23803	GP
COONEY, MD, Dorothy F. Post Office Box 816 Farmville, VA 23901	R	HAMNER, MD, James L Mannboro VA 23105	GP	MASON JR., MD, James D 507 S Sycamore St Petersburg VA 23803	IM	RUHNKE JR, MD, Edward E 530 S Sycamore St Petersburg VA 23803	OBG	WEATHINGTON II, MD, Lee 815 W Poythress St Hopewell VA 23860	FP
COPPEDGE, MD, Chas Wm 205 N Virginia St Farmville VA 23901	GP	HANEY, MD, Ronald L. P O Box 466 Farmville, VA 23901	ORS	MASRI, MD, Asad M 511 S Sycamore St Petersburg VA 23803	P	RUSSI, MD, Simon 101 Queen Street Alexandria, VA 22314	PTH	WEBB JR., MD, Robt B 408 S Sycamore St Petersburg VA 23803	IM
CROISIER, MD, Joseph L P O Box 1835 Petersburg VA 23803	ORS	HARGROVE JR, MD, Roy Belmont Box 247 Farmville VA 23901	GS	MC ILWAINE III, MD, Wm B 434 W Washington St Petersburg VA 23803	PD	SADIGHIAN, MD, Z Dean Box 427 South Hill VA 23970	GP	WHITE, MD, Stuart Bruce 820 S Main Blackstone VA 23824	FP
CROWDER JR., MD, Chas H 212 Mecklenburg Ave South Hill VA 23970	FP	HARRIS JR, MD, Andrew E 820 So Main St Blackstone VA 23824	FP	MCCLURE, MD, Phillip H 2115 Dodson Road Petersburg, VA 23805	R	SARAYBA, MD, Alberto A 501 North 6th Ave Hopewell, VA 23834	GS	WHITTLE, MD, Joseph P. Box 339 Colonial Heights VA 23834	IM
CROWDER, MD, Margaret E 444 Halifax St Petersburg VA 23803	IM	HARRIS, MD, James Selden 820 So Main St Blackstone VA 23824	GP	MCILWAINE, MD, Benjamin H. Route 1, Box 16 Sutherland VA 23885	US	SAUNDERS, MD, Thos Archer Box 396 South Hill VA 23970	GP	WILLIAMS II, MD, W.C. P O Box 586 Farmville, VA 23901	FP
DANIELS, MD, Warren C. P O Box 831 Hopewell, VA 23860	IM	HARRY, MD, Frederick P. P O Box 339 Colonial Heights, VA 23834	IM	MENENDEZ III, MD, Reinaldo 507 Laurel St Emporia VA 23847	GS	SCHWARTZ, MD, Julius L 208 South Sycamore St Petersburg VA 23803	D	WILLIAMS, MD, Mark Byrd 12 Hillcrest Road Petersburg VA 23805	GP
DAW, MD, Albert Lee Post Office Box 98 South Hill, VA 23970	GS	HART JR., MD, Kirby Thompson 111 Morton Ave Petersburg VA 23805	PD	MILLER, MD, Edith I Bena VA 23018	TR	SEPDHAM JR., MD, Taweesuk 14505 Walthall Drive Colonial Heights, VA 23834	N	WORARATANADHARM, MD, S 116 Beech Drive Hopewell VA 23860	OBG
DOSHI, MD, Ramesh D. 6437 Moon Lane Richmond, VA 23234	R	HAUSER, MD, Walter Arthur 1947 Walton Street Petersburg VA 23803	CHP	MILLER, MD, Ronald Edward 815 W Poythress Hopewell VA 23860	FP	SETJIRAVIROJ JR., MD, Prasert 6 Doctors Drive Emporia VA 23847	IM	YATES, MD, Munford Radford 507 S Sycamore St Petersburg VA 23803	IM
DOUGHERTY, MD, Clyde Hudson 303 Appomattox St Hopewell VA 23860	GP	HEDRA JR., MD, Tibor 8 Marshall St Petersburg VA 23803	US	MOORE JR., MD, De Saussure P 815 W Poythress St Hopewell VA 23860	GP	SHELTON, MD, Wm A Box 237 Boydton VA 23917	GP		
		HERRING JR., MD, Alvah L 6606 Park Avenue Richmond, VA 23226	GS	MOORE, MD, Dorothy Diehl 2032 Mattoax Ave W Petersburg VA 23803	GP				

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YEH III, MD, Li-Cheng P O Box 2145 Petersburg VA 23803	AN	CHITWOOD, MD, Sarah E Roberts R F D 1, Box 250 Draper, VA 24324	P	GILLET JR, MD, Richard C. P O Box 2867 Radford, VA 24143	FP	KING, MD, James P 502 Seventh St Radford, VA 24141	P	NUCKOLS, MD, Wm Andy 227 West Main Street Abingdon VA 24210	FP
YOUNG, MD, Estelle Irene 612 A South Sycamore Petersburg, VA 23803	D	CHITWOOD, MD, Walter R Wytheville VA 24382	OS	GILMER, MD, Giles O Lebanon Medical Group Lebanon VA 24266	GP	KING, MD, James Peter 104 Doctors Park Galax, VA 24333	OTO	PARK, MD, Chas Lieben 700 Church St Blacksburg VA 24060	IM
YOUNGBLOOD, MD, Walker P Box 480 Hopewell VA 23860	GP	CHRISTIAN, MD, Wm E 34 Ridgefield Lane Radford VA 24141	AN	GILMER, MD, Robert D. Box 38 Abingdon, VA 24210	AN	KING, MD, Mariano L. Russell County Medical Center Lebanon VA 24266		PATEL, MD, Bharat R. P O Box 3636 Radford, VA 24143	DR
ZUCKERMAN, MD, Ellis N Box 710 Petersburg VA 23803	IM	CHUNG III, MD, Jay-Dea 707 Randolph St Radford VA 24141	AN	GINTHER, MD, Jeffrey Paul 101 North Street Bristol, VA 24201	FP	KING, MD, Wm Whitman 707 Randolph St Radford VA 24141	U	PATTERSON JR, MD, James L 25 Fourth St Nw Pulaski VA 24301	FP
<b>SOUTHWEST VIRGINIA</b>									
ALDERFER, MD, Richard D Route 2, Box 539 Radford VA 24141	GP	CONANT, MD, Roger 307-A Hernando St Ft Pierce, FL 33449	GP	GLOVER, MD, Roger Arthur P O Box 596 Abingdon VA 24210	OBG	KNARR, MD, John Weidner 810 Prospect Avenue Pulaski VA 24301	IM	PATTERSON, MD, James Edwin 1154 Snider St Marion VA 24354	FP
AMONETTE, MD, Wilbur F Box 3746 Radford VA 24143	EM	CONRAD, MD, Frederick Ellison 211 W Main St Abingdon VA 24210	OPH	GRAHAM, MD, Chas Mc Donald Box 1087 Radford VA 24141	OBG	LANGEBECK, MD, Miguel 306 Little Circle Blacksburg VA 24060	PTH	PECK, MD, Jos C 199 Hospital Drive Galax VA 24333	GP
ARMBRISTER, MD, Douglas K Radio Road Marion VA 24354	GS	COOK JR, MD, William Henry 2 Pine Tree Lane Radford, VA 24141	PD	GRANTHAM, MD, Alan W Johnston Memorial Clinic Abingdon VA 24210	GS	LARSEN, MD, Geoffrey Arthur 161 Stonewall Heights Abingdon VA 24210	GS	PEREZ, MD, Ana Maria 14 Holly Lane Radford VA 24141	CLP
AUSTIN, MD, Chas N 210 Stonewall Heights Box 308 Abingdon VA 24210	R	COX, MD, James Glenn Box 67 Hillsville VA 24343	GP	GRAYBEAL, MD, Henry Charlton 785 Mountain View Dr Christiansburg VA 24073	GP	LEBITA-CEBALLOS, MD, Victoria M 297 Panorama Dr Marion VA 24354	PD	PEREZ, MD, Antonio 14 Holly Lane Radford VA 24141	PTH
BAILEY, MD, Harloe Box 235 Rural Retreat VA 24368	GP	COX, MD, Virgil Jefferson 101 N Monroe Street Galax VA 24333	GP	GREEVER, MD, Donald L P O Box 346 Chilhowie VA 24319	IM	LEE, MD, John Edward P O Box J Pearisburg, VA 24134	OBG	PETTIGREW, MD, James Andrew 3-P Doctor's Bldg Bristol TN 37620	NS
BARRANCO, MD, S. D. 3708 S Main St Ste A Blacksburg VA 24060	ORS	CRANTON, MD, Elmer Mitchell Mt Rogers Clinic, Box 44 Trout Dale VA 24378	FP	GREEVER, MD, Wm N P O Box 346 Chilhowie VA 24319	GP	LEE, MD, Thaddeus Carmichael 600 Randolph Street Radford VA 24141	GS	PHLEGAR, MD, David Shanks 505 Airport Rd Sw Blacksburg VA 24060	GP
BARROW, MD, Guy Jos Johnston Mem Clinic Abingdon VA 24210	IM	CUBE, MD, Ernesto Milla Box 928 Radford VA 24141	US	GUINTER, MD, Robert H Med Arts Bldg, Radio Hill Rd Marion VA 24354	PD	LEE, MD, Thaddeus Carmichael 600 Randolph Street Radford VA 24141	FP	PINKERTON JR, MD, Herman H Johnston Mem Clinic Abingdon VA 24210	PDA
BARTA JR, MD, Joseph A P O Box 1172 Radford VA 24141	OPH	DAVIE, MD, Steven Ames Medical Arts Bldg Blacksburg VA 24060	OTO	HAAS, MD, Theron Henry 200 8th St Box 572 Radford VA 24141	OBG	LEVY, MD, Jan Alfred 117 Third Ave Radford VA 24141	GS	PORTER, MD, Walter A Box 5 Hillsville VA 24343	GP
BARTON, MD, Walter Seignious P O Box 642 Wytheville VA 24382	GP	DAVIS JR, MD, Russell Lewis 600 Randolph St Radford VA 24141	FP	HALL JR, MD, Glenn Claire P O Box 1206 Radford VA 24141	PD	LUCK, MD, James Thos Box 456 Damascus VA 24236	FP	OUNIN, MD, Karen L. Alleghany Clinic Shawsville, VA 24162	FP
BASSHAM, MD, Harold Lee P O Box 866 Abingdon VA 24210	R	DAVIS, MD, Chas Young Montgomery County Com Hosp Blacksburg VA 24060	OBG	HATFIELD, MD, Cecil C Drawer C C Saltville VA 24370	GP	LUTTRELL, MD, Homer B 907 Prospect Avenue Pulaski VA 24301	GP	REPASS, MD, Robt A Four Winds Bristol VA 24201	GS
BISHOP, MD, Wiffiam D. 106 Wadsworth Street Radford VA 24141	OTO	DAVIS, MD, Thos Philip P O Box 846 Christiansburg VA 24073	GP	HATFIELD, MD, Wm Henry 701 Preston Ave, Sw Blacksburg, VA 24060	PH	MACKLER, MD, Stuart F P O Box 3768 Radford, VA 24143	ORS	RIOS, MD, Juan Francisco 199 Hospital Drive Galax VA 24333	GS
BLALOCK, MD, Jos Rogers 657 Holston St Marion VA 24354	P	DEANS, MD, Robt Douglas 906 Mc Bryde Dr Blacksburg VA 24060	U	HAWKINS JR, MD, Richard F. 109 Spottswood Place Marion, VA 24354	R	MALIN, MD, Wendell Eugene Rt 2 Wytheville VA 24382	ABS	ROBINETTE, MD, Emory H. Johnston Memorial Clinic Abingdon, VA 24210	PD
BLANTON JR, MD, Frank 350 Blountville Hwy, #201 Bristol TN 37620	GS	DRYSDALE, MD, Daniel B Medical Arts Bldg Blacksburg VA 24060	OPH	HENDRICKS JR, MD, Wm Tillman 200 Counrty Club Drive Blacksburg VA 24060	FP	MANALO JR, MD, Buenaventura Cafawba Hospital Cafawba, VA 24070	IM	ROEBUCK, MD, Basil Enoch P O Box 49 Blacksburg, VA 24060	P
BLATTNER, MD, Carlos Luis Box 616 Marion, VA 24354	IM	DUNMAN, MD, Lester Edwin 1519 Cabot Drive Pearisburg VA 24134	IM	HENDRIX, MD, Paul C 655 East Pine Street Wytheville VA 24382	IM	MANDELSTAMM, MD, Maria T 600 Lendsdowne Blacksburg VA 24060	IM	ROGNEY, MD, Douglas Lloyd 710 W Ridge Rd Wytheville VA 24382	FP
BLOSE, MD, Donald Curtis 199 Hospital Drive Galax VA 24333	GS	DYER, MD, Raymond Douglas Plaza 1 200 Country Club Dr Blacksburg VA 24060	GP	HORNEY, MD, Wayne D. Box 718 Wytheville, VA 24382	FP	MARTIN, MD, Morr Glenwood Box 576 Hillsville VA 24343	FP	RUST, MD, John Newton Route 2, Box 93 Rural Retreat, VA 24368	PTH
BOATWRIGHT, MD, Chas Lee 303 Church St Blacksburg VA 24060	GP	EARLY JR, MD, Jos H 160 Main, P O Box 37 Hillsville VA 24343	GP	HORNSBY, MD, Robert P. 151 Hillside Drive Abingdon, VA 24210	A	MC CLANE, MD, John Raymond Route 2, Box 63-B Wytheville, VA 24382	R	RYPLANSKY, MD, Anatol P O 1586 Dublin, VA 24084	R
BOLEN, MD, John Wm 199 Hospital Drive Galax VA 24333	OBG	EDEEN JR, MD, James Arthur Route 1, Box 162 Pembroke, VA 24136	P	HORSCH, MD, Robert F. Lebanon Medical Group Lebanon, VA 24266	GS	MC DOWELL, MD, Alice W. 630 Hill Top Street Marion, VA 24354	PD	SCHIFFERT, MD, Chas Wilson V P I Student Health Services Blacksburg VA 24061	GP
BOLTER, MD, Delano Woodrow 416 East Main Street Marion, VA 24354	P	ELLER, MD, Jos Johnson 233 Holly Street Marion VA 24354	FP	HUGHES, MD, Chas B Box 327 Wytheville VA 24382	PD	MC GUIRE, MD, Erma J Marra 705 Wenonah Avenue Pearisburg VA 24134	GP	SCHMIDT, MD, Edmund Julian P O Box 810 Blacksburg VA 24060	GP
BONIFACE JR, MD, John 199 Hospital Dr Ste 5 Galax VA 24333	IM	ELLIOTT, MD, James Wm Lebanon VA 24266	GP	HULVEY, MD, J Thomas 300 East Valley Street Abingdon VA 24210	ORS	MC KAIN, MD, Carey W. 300 E Valley Street Abingdon, VA 24210	ORS	SHAFFER, MD, John S 227 West Main St Abingdon VA 24210	GP
BOOKER III, MD, James Judson P O Box 642 Wytheville VA 24382	FP	ELLIOTT, MD, Walter C Lebanon VA 24266	GP	HYLTON, MD, James Moir 25 4th St Pulaski VA 24301	GP	MEINCKE, MD, David Lee Montgomery County Hospital Blacksburg VA 24060	OBG	SHOWALTER, MD, Josiah Thos 10 Hickok St Christiansburg VA 24073	GP
BORILLO, MD, Romeo Bisquera P O Box 419 Pearisburg, VA 24124	GP	ELSWICK, MD, Ronald Kenneth Box 612 Radford VA 24141	OBG	IRVIN, MD, Emory R P O Box 48 Blacksburg VA 24060	GP	MERKER, MD, Frank F Post Office Box 30 Troutdale, VA 24378	OS	SKEWES, MD, David Jessop P O Box 638 Dublin VA 24084	GP
BOWDEN, MD, James Harris Johnston Memorial Clinic Abingdon VA 24210	IM	FANT, MD, Palmer Willis Rt 1 Independence VA 24348	R	ISENHOUR, MD, Wm Apperson 3708 South Main Street Blacksburg VA 24060	OBG	MICHAEL, MD, Carlton A 706 Preston Ave Blacksburg VA 24060	OS	SLAYTON, MD, Michael Edward Medical Arts Bldg-Rt 460 South Blacksburg VA 24060	IM
BRILLHART, MD, David M 227 W Main St Abingdon VA 24210	FP	FAUSTINO, MD, Benigno D Cedar Valley Apts, #900-B Radford, VA 24141	CLP	JEAN, MD, Cheo Ming Radford Community Hospital Radford, VA 24141	EM	MILLER, MD, Calvin Lewis Box 126 160 Valley St, Ne Abingdon VA 24210	OPH	SMITH JR, MD, Geo Robt Shawsville VA 24162	FP
BULLOCK, MD, Richard Edward V P I Student Health Ctr Blacksburg VA 24061	OS	FINN, MD, Rolfe Baxter P O Box 3608 Radford, VA 24143	P	JONES, MD, George Frederick Med Arts Bldg, Radio Hill Rd Marion VA 24354	PD	MISTR, MD, Ernest Noel Johnston Memorial Clinic Abingdon VA 24210	PD	SMITH JR, MD, Oscar Orton Radio Rd Marion VA 24354	ABS
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CALILUNG, MD, Cesar S 400 Main St Narrows VA 24124	GS	GAILLIOT, MD, Robert Vernon Route 5, Box 192 Marion VA 24354	CLP	KECK, MD, Wm D Box 3585 Radford VA 24143	P	MORGAN, MD, Snead Wesley Johnston Memorial Clinic Abingdon VA 24210	GS	SPENCER, MD, Harrison C Johnston Memorial Clinic Abingdon VA 24210	PD
CATRON JR, MD, Stuart H 191 Johnston St Abingdon VA 24210	IM	GARDNER, MD, James L 300 E Valley St Abingdon VA 24210	ORS	KEGLEY JR, MD, James B Drawer V Saltville VA 24370	PH	MOSKOWITZ, MD, Edward J. 1319 North Main Street Marion, VA 24354	U	SPILLMAN, MD, James Blair Box 1206 Radford VA 24141	PD
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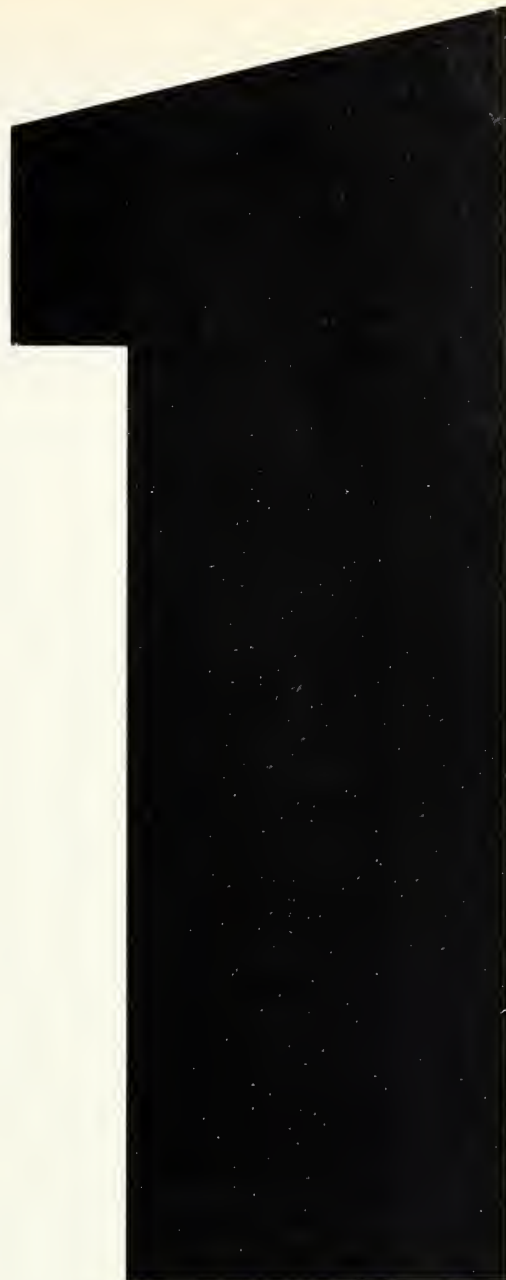
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DEVLIN, MD, James Timothy 1120 First Colonial Rd Virginia Beach, VA 23454	FP	HAMILTON, MD, Colin W 1000 1st Colonial Rd, #1 Virginia Beach, VA 23454	ORS	MARVEN, MD, Lee Jonathan 4512 Old English Cir Virginia Beach, VA 23455	EM	ROBERTSON, MD, Robt John 1169 First Colonial Rd #5 Virginia Beach, VA 23454	CD	TROIANO, MD, Raymond G 1008 1st Colonial Rd, #101 Virginia Beach, VA 23454	N
DEWALT JR., MD, Chester W 1728 Sir William Osler Dr Virginia Beach, VA 23454	GP	HARMATUK, MD, Frances A. 4620 Haygood Road, #5 Virginia Beach, VA 23455	P	MASCARINAS JR., MD, Teofilo C 4551 Professional Circle Virginia Beach, VA 23455	IM	RODGERS, MD, Stephen Quarlas 1744 Sir William Osler Dr Virginia Beach, VA 23454	PD	VINH, MD, Luc 4656 Haygood Rd, #D Virginia Beach, VA 23455	FP
DICKINSON, MD, Wm Andrew 1060 First Colonial Rd Virginia Beach, VA 23454	CD	HARRIS, MD, Edward David 2409 Spinnaker Ct Virginia Beach, VA 23451	GP	MASON, MD, Gordon L. Bayside Medical Plaza #2B Virginia Beach, VA 23455	OPH	RUSS JR., MD, Clarke 1016 First Colonial Rd Virginia Beach, VA 23454	ORS	WADDELL, MD, Robert W 1100 First Colonial Rd Virginia Beach, VA 23454	ORS
DILLON, MD, James D 1229 East Bay Shore Dr Virginia Beach, VA 23451	NS	HARRISON, MD, H. Courtenay 1008 1st Colonial Rd, #103 Virginia Beach, VA 23454	END	MATCHETT, MD, Robert M. 1000 First Colonial Rd Virginia Beach, VA 23454	AN	SADLER JR, MD, Charles R. 1100 First Colonial Rd Virginia Beach, VA 23454	ORS	WALLACE JR., MD, Karl Kenneth 1309 North Bay Shore Dr Virginia Beach, VA 23451	DR
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DOWLING, MD, Jean Marie 1120 1st Colonial Rd, #200 Virginia Beach, VA 23454	CHP	HILL JR., MD, Trafford 1020 First Colonial Rd Virginia Beach, VA 23454	P	MC CUNE, MD, Frederick K 1000 First Colonial Rd Virginia Beach, VA 23454	AN	SANTIAGO, MD, Gloria A. 4551 Professional Circle Virginia Beach, VA 23455	OBG	WARREN, MD, Steven S. 1000 1st Colonial Rd, #101 Virginia Beach, VA 23454	U
DOWNS, MD, Peter Ellsworth Bayside Hosp, Emerg Dept Virginia Beach, VA 23455	EM	HILL, MD, Kathryn Dadds 228 N Lynnhaven Rd Ste 130 Virginia Beach, VA 23452	PD	MC DANIEL, MD, David H. 1615 Colonial Medical Ct Virginia Beach, VA 23454	D	SARRETT, MD, David Lee 915 First Colonial Rd Virginia Beach, VA 23454	OBG	WARDEN, MD, Wm Budd 2017 Pleasure House Rd Virginia Beach, VA 23455	GP
DUNDON, MD, Bruce Carroll 816 Independence Blvd, #2-H Virginia Beach, VA 23455	D	HOLLAND, MD, Clarence Adrian 2017 Pleasure House Rd Virginia Beach, VA 23455	GP	MEADE JR., MD, Thomas S. 1100 First Colonial Road Suite 101 Virginia Beach, VA 23454	ORS	SAUNDERS JR., MD, Milton A Pembroke Six, Suite 129 Virginia Beach, VA 23462	D	WARREN, MD, Bertram Lee 1004 1st Colonial Rd, #101 Virginia Beach, VA 23454	OBG
DUNNINGTON, MD, Arthur R 1012 First Colonial Rd #103 Virginia Beach, VA 23454	RHU	HUDSON, MD, Sue Beth 2380 Bays Edge Ave Virginia Beach, VA 23451	IM	MILLER, MD, Mitchell B 1776-D Princess Anne Rd Virginia Beach, VA 23456	FP	SCHNEIDER, MD, Stephen A 453 Edwin Drive, #101 Virginia Beach, VA 23462	GS	WARTH, MD, Gregory James 1860 Colonial Medical Court Virginia Beach, VA 23454	IM
EAGLES, MD, Donald Taylor 1120 First Colonial Rd Ste 204 Virginia Beach, VA 23454	OTO	HUGHES, MD, Felix Austin 225 63rd Street Virginia Beach, VA 23451	DR	MILLER, MD, Ira David #3, Independence Med Cen Virginia Beach, VA 23455	GS	SCHICK, MD, Eduardo Jorge 3420 Holland Rd, #104 Virginia Beach, VA 23456	OBG	WASHBURN, MD, Ronald Lee 1632 Bay Point Drive Virginia Beach, VA 23454	R
EAMES, MD, Bruce Lee 1320 Baycliff Drive Virginia Beach, VA 23454	OTO	IGLECIA, MD, Raymond P P O Box 62243 Virginia Beach, VA 23462	P	MILLER, MD, Murray Culbertson 3191 Adam Keeling Rd Virginia Beach, VA 23454	FP	SCHREIBER, MD, Douglas R. 1100 First Colonial Rd Virginia Beach, VA 23454	ORS	WASSERMAN JR., MD, L Leslie 1004 1st Colonial Rd #101 Virginia Beach, VA 23454	OBG
EARLEY JR., MD, Chas Marion P O Box 4159 Virginia Beach, VA 23454	GS	IMBUR, MD, Donald James 1856-B Colonial Med Court Virginia Beach, VA 23454	U	MIRZAK, MD, John Arnold 1020 First Colonial Rd Virginia Beach, VA 23454	P	SCHREINER, MD, Carol Anne 1387 Little Neck Road Virginia Beach, VA 23452	P	WEST, MD, Richard Duane 1856 Colonial Medical Ct Virginia Beach, VA 23454	GS
EASTON, MD, Richard E. Va. Beach Municipal Ctr. Virginia Beach, VA 23456	IM	JARRETT, MD, Alvin Quarles 4704 Ocean Front Virginia Beach, VA 23451	AN			SCHWARTZ, MD, Arnold Jay 453 Edwin Dr, #102 Virginia Beach, VA 23462	D	WHITE, MD, Julian Andrews P O Box 825 Virginia Beach, VA 23451	FP

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## 34 VIRGINIA BEACH—WILLIAMSBURG—WISE—RESIDENTS

WHITE, MD, Rolfe Downing 913 North Plantation Dr Virginia Beach, VA 23454	OBG *	HENDERSON, MD, Clifford E 502 Strawberry Plains Rd Williamsburg VA 23185	FP	WISE	PHILLIPS JR, MD, Joseph T. Virginia Ave-10th St Norton VA 24273	GS *	BROWN, MD, Kimberley L 2924 Princess Anne Cres Chesapeake, VA 23321	PTH
WHITNEY, MD, Hugh Raymond H 1000 First Colonial Rd Virginia Beach VA 23454	AN *	HENDERSON, MD, June Rose S 119 Dover Rd Williamsburg VA 23185	FP	AWAN, MD, Khalid Javed 1921 Park Ave.S.W Norton VA 24273	OPH *	GP *	BROWN, MD, Loretta P 260 James River Dr Newport News, VA 23601	IM
WILLIAMS, MD, David L. 3386 Holland Rd, #205 Virginia Beach, VA 23452	OBG	HESS, MD, James Brock 105 Crestwood Drive Williamsburg VA 23185	IM	BALASUBRAMANIAM III, MD, M Big Stone Clinic Big Stone Gap VA 24219	PD	R	BUNCH III, MD, Morgan R 2500 Pocoshock Place Richmond, VA 23235	FP
WILLIAMS, MD, Frederick M 1120 First Colonial Rd Virginia Beach VA 23454	OPH *	HOFFMIER, MD, Thomas Joseph 1006 Richmond Road Williamsburg, VA 23185	IM	BARTON, MD, Wm Baynard P O Box 6 Stonega VA 24285	OM	R	BURTON, MD, Ashby Jeffries 610 Citizens Trust Bldg Portsmouth, VA 23704	FP
WILLIAMS, MD, James Newton 717 Cardinal Rd Virginia Beach VA 23451	P *	HOWARD, MD, Hall Renfro 1115-A Old Colony Lane Williamsburg, VA 23185	GS	BAUSCH, MD, Robert Stephen Box 17 Wise VA 24293	FP	PD *	CALIHAN, MD, Martha S. 4223 S Four Mile Run Dr, #22 Arlington, VA 22204	FP
WILLIAMSON, MD, John A. 1100 First Colonial Road Virginia Beach, VA 23454	ORS *	INLOES JR, MD, Benj H 70 Winstler Fax Williamsburg VA 23185	OBG	CAPALAD, MD, Elpidio Fajardo 100 15th Street NW Norton VA 24273	GP *	GS	CAMOMOT-WITTEN, MD, Vilma 3136 Valentino Court Oakton, VA 22124	FP
WONG, MD, George Sonny 2017 Pleasure House Rd Virginia Beach, VA 23455	FP *	JENKINS, MD, Daniel 128 Tolars Road Williamsburg VA 23185	OBG	CARTAGENA, MD, Rodolfo S P O Box 727 Norton, VA 24273	OBG	IM	CAMPBELL, MD, Kevin M 2508 Kenmore Rd South Richmond, VA 23225	PD
WRIGHT, MD, James Claude 1120 1st Colonial Rd Virginia Beach VA 23454	OPH	JEWUSIAK, MD, Edward M 1138 Professional Dr Williamsburg VA 23185	ORS	CHEUNG, MD, Chin Lin 381 Suncrest Drive Bristol VA 24201	EM	FP	CARRICO, MD, Thomas J Box 154, M C V Station Richmond, VA 23298	PS
WYLES, MD, Ronald Jos 1200 1st Colonial Rd, #200-M Virginia Beach VA 23454	D *	JONES, MD, Joseph L 135 Second Street Williamsburg VA 23185	GP *	CROSS, MD, James Allerton Hanging Rock Clinic, Drawer T St Paul, VA 24283	GP *	GS	CAULKINS, MD, Michael K 112-A Windsor Castle Dr Newport News, VA 23602	FP
YEH, MD, Betty P-Y 770 Independence Circle Suite 101 Virginia Beach, VA 23455	IM *	JONES, MD, Roger Wm 1115 Professional Drive Williamsburg VA 23185	OBG	DAVIS, MD, William Alvin Hanging Rock Clinic, Drawer T St Paul VA 24283	GP *	U *	CAULKINS, MD, Pamela M 112-A Windsor Castle Dr Newport News, VA 23602	FP
YI, MD, Chu Hon 1821 Old Donation Pkwy, #4 Virginia Beach VA 23454	IM *	KAISER, MD, John R. 1304-A Mt Vernon Ave Williamsburg, VA 23185	IM	FLEENOR, MD, Lawrence J Route #1, Box 468 Big Stone Gap VA 24219	FP	GP	CHITNUM, MD, Susan L. 712 Raleigh Ave Norfolk, VA 23507	OBG
WILLIAMSBURG								
ADAMS, MD, Kenneth Atwell The Professional Bldg Williamsburg, VA 23185	GE	KAROW, MD, Juliette Seelye Stud Health Serv, W & M Colleg Williamsburg VA 23185	GP	FORD, MD, Michael B Big Stone Gap Clinic Big Stone Gap VA 24219	GP	PD *	CLARKE, MD, Robert W. P O Box 1075 Franklin, VA 23851	EM
BARTON, MD, James Edwin 706 Jamestown Road Williamsburg VA 23185	FP	KIMBROUGH, MD, Janet Coleman Tucker House Williamsburg VA 23185	GP *	FOSTER, MD, Glen G 406 Washington St Gallax VA 24333	FP *	DR *	COLEMAN, MD, Peter R. 820 Main Street Blackstone, VA 23824	FP
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BLAYTON, MD, James Blaine 150 Ron Springs Drive Williamsburg VA 23185	GP *	MAZUR, MD, Stephen H 107 West Kingswood Dr Williamsburg VA 23185	AN *	GONZALEZ, MD, Ulysses S 100 15th Street, Nw Norton VA 24273	IM	R	CROSS, MD, Steven W 2310 Chancellor Road Richmond, VA 23235	CD
BORLAND, MD, David S 1238 Mount Vernon Ave Williamsburg VA 23185	PTH *	MUSGRAVE, MD, Joseph Walker 138 John Tyler Highway Williamsburg VA 23185	D	HASHEM, MD, Abul P O Box 1800 Wise, VA 24293	R	FP	DANIEL, MD, John G 201 College Ave Blackstone, VA 23824	FP
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BUNTING, MD, Richard Fry 224 Monticello Ave Williamsburg VA 23185	OPH *	PACKER, MD, Gerald A. 108 Godspeed Lane Williamsburg VA 23185	EM	INGRAM, MD, Ernagene F P O Box 187 Norton VA 24273	PH	PD *	DI GIOVANNI JR, MD, Cleto 11091 Saffold Way Reslon, VA 22090	P
BURDICK, MD, Edward Peter 454-A Wythe Creek Rd Poquoson VA 23662	GP	PARMELEE, MD, Dean X 642 Counselsors' Way Williamsburg, VA 23185	CHP	INGRAM, MD, Lewis Karl P O Box 187 Norton VA 24273	OBG	OBG	DIAZ, MD, Dennis D. 1774 Chase Arbor Common Virginia Beach, VA 23462	GS
CHOHANY, MD, Geo J 230 Monticello Ave Williamsburg VA 23185	GS *	PERRY, MD, William J Student Health Center Williamsburg, VA 23185	FP	JAIN, MD, Sureschand D Box 17 Wise VA 24293	PD	GE	DIXON, MD, James G 26 South Greenfield Ave Hampton, VA 23666	FP
CILLEY, MD, Richard D 139 Tanbark Lane Williamsburg VA 23185	OS	PHARR III, MD, Scott Yorke 504 Strawberry Plains Rd Williamsburg VA 23185	OTO	JONES, MD, Delmas Bernard Box 1039 Wise VA 24293	R *	DR	DORSEY III, MD, John Thomas Portsmouth Naval Hospital Portsmouth, VA 23708	IM
CUMMINGS, MD, Wm Steven 124 Tanbark Lane Williamsburg VA 23185	FP	PITMAN, MD, John Mathews 326 Monticello Ave Williamsburg VA 23185	GS *	KANWAL, MD, Gurcharan Singh P O Box 1136 Coeburn VA 24230	IM *	FP *	DRAKE JR, MD, Charles R. 230 West Boscawen St Winchester, VA 22601	EN
DIMMETT, MD, James David P O Box 253 Williamsburg VA 23187	CHP *	POLESIC III, MD, Slobodan 117 Dover Rd Williamsburg VA 23185	PTH	KESSLER, MD, Wm A Box 17 Wise VA 24293	GS	GS	DUNWIDDIE, MD, Walter C. 505 Harrow Road Richmond, VA 23225	AN *
EBERDT, MD, Arthur Jackson 1306 Mount Vernon Avenue Williamsburg VA 23185	OPH	POWERS JR, MD, Thos Jefferson 502 Strawberry Plains Road Williamsburg VA 23185	FP	KOTAY, MD, S.C. Box 17 Wise, VA 24293	ORS *	OBG	ELLER JR, MD, M Edward Box 98, Uni Va Med Ctr Charlottesville, VA 22908	FP
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FLETCHER, MD, John Stevenson 531 Mill Neck Rd Williamsburg VA 23185	PD	ROSENBERG, MD, Maurice S. 212 John Ratcliffe Williamsburg, VA 23185	GP *	LITTON, MD, Frederick Mitchel Big Stone Gap Clinic Big Stone Gap VA 24219	GP	OBG	ERICKSON, MD, Reed A 3278 Sydenham St. Fairfax, VA 22031	FP
GARCIA, MD, Leoncio Antonio 304 Lockley Drive Williamsburg VA 23185	P	SANZ, MD, Manuel O 43 Winstler Fax Williamsburg VA 23185	P	LUTHRA, MD, Ramesh C P O Box 799 Wise, VA 24293	U	IM	ESCHENROEDER JR, MD, H.C. Route 5, Box 228 Charlottesville, VA 22901	ORS
GERDE, MD, Leland 111 Albemarle Dr Williamsburg, VA 23185	OPH	SIM, MD, Peter Alan 107 Winstler Fax Williamsburg VA 23185	EM	MAINE, MD, Charles P. Wise Clinic, Box 17 Wise, VA 24293	IM *	NS	EVANS, MD, Toni I. 2311 Seagull Ct Richmond, VA 23229	IM
GRAHAM, MD, Maurice E 1101 Prof Drive, #D Williamsburg, VA 23185	PD	SPIRN, MD, Camilla B. 132 John Tyler Highway Williamsburg, VA 23185	OBG	MANOHARAN, MD, E Big Stone Gap Clinic Big Stone Gap VA 24219	OBG	FP	EVERETT, MD, Joel C. 908-B Gaskins Road Richmond, VA 23233	OBG
GRASINGER, MD, John E 1138 Professional Dr Williamsburg, VA 23185	ORS	STEIGER, MD, Wm Anthony 225 West Tazewell Williamsburg, VA 23185	IM *	MAPHIS JR, MD, Frederick D P O Box 176 Wise, VA 24293	PD	FP	FAUZIA, MD, Mutahar 1714 N Troy St, #798 Arlington, VA 22201	FP
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HAMRICK, MD, Robt Arnold Box 1846 Williamsburg VA 23185	GER *	TAYLOR, MD, Anthony J P O Box 254 Williamsburg VA 23187	U	MURTHY, MD, H.R. Rama Dickenson Wise, Box 17 Wise, VA 24293	OBG *	IM	FOSSEY, MD, Mark D. 805 Ward Ave Charlottesville, VA 22901	P
HANGER, MD, Keith Elwood 1006 Richmond Rd Williamsburg VA 23185	IM *	THEIS, MD, Richard Braxton 502 Strawberry Plains Rd Williamsburg VA 23185	FP	NAKANDAKARI, MD, Masao Box 17 Wise VA 24293	GP	ON	FOSTER, MD, Helen M. 4120 Ketchum Dr. Chesterfield, VA 23832	OBG
HELVIG, MD, Warren Bowman General Delivery Zanoni, VA 23191	PTH	THIEL, MD, Martin August 1310 Mt Vernon Ave, #B Williamsburg VA 23185	GS *	NELSON, MD, Pierce Danl Mental Health Clinic Box 920 Wise VA 24293	P *	FP	FRAGA, MD, Vivian Maria 4518 Seminary Road Alexandria, VA 22304	GS
		VAN DRIEM, MD, Geo Henri P O Box 669 Toano VA 23168	GP *	NORTH, MD, Arthur W. P O Box 17 Wise, VA 24293	OBG	FP	FRANCO, MD, Kenneth L. 4518 Raleigh Ave Alexandria, VA 22304	FP
				PARANTHAMAN, MD, S. K. 107 East Main Street Appalachia, VA 24216	PUD *	FP	FRONC JR, MD, Henry Conrad 103 Second Street South Hill, VA 23970	IM

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FUNKHOUSER, MD, Laura S. 4811 Park Avenue Richmond, VA 23226	PM	LUDEMAN JR, MD, Douglas H. 4404 Park Avenue Richmond, VA 23221	U	SINGER, MD, Jerry R. 5400 Bernard Drive Sw Roanoke, VA 24014	IM	ANAMA, MD, Emmanuel Desling 227 Commercial Ave Fulton, KY 42041	AN	CRIDER, MD, Donald Bryant 501 Howard Ave Altone PA 16601	P
GARDNER, MD, Stephen R. 10625 March Hare Dr Richmond, VA 23235	NS	MALCHOFF, MD, Carl D. 2644 Jefferson Park Cir Charlottesville, VA 22903	EN	SMITH, MD, Richard A. 9 Gale Circle Newport News, VA 23606	FP	ANGELL, MD, Franklin Lynwood 645 Lakeland S Severna Park MD 21146	R	DAFASHY, MD, Mounir Y 2264 Eisenhower Riverside CA 92506	P
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GELFMAN, MD, Daniel M. 11610 Timberly Waye Richmond, VA 23233	IM	MARSH, MD, John O. P O Box 3 Strasburg, VA 22657	IM	SOULE, MD, John Phillip 2212 E Tremont Richmond, VA 23225	IM	ARTMAN, MD, Ralph T P O Box 854 Bendon OR 97411	U	DAVIS, MD, Arthur A 8200 16th St, NW Washington DC 20012	FP
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GRATHWOHL, MD, Mark A. 2510 West Tremont Ct Richmond, VA 23225	PTH	MERKEL, MD, Victoria Lessinger 380 Maple Avenue Vienna, VA 22180	FP	STEC, MD, Rita Jeanne 1441 Moultrie Ave, #3 Norfolk, VA 23509	ON	AUERBACH, MD, M. Richard 401 Nw 42nd Street Plantation, FL 33317	PD	DEMIRAY, MD, Adel 801 Toll House Rd Frederick MD 21701	GS
GRIFFIN, MD, Gary K. 243 Shamrock Road Charlottesville, VA 22903	R	MILLER, MD, Thomas J. 2223 Hanover Ave Richmond, VA 23220	IM	STEFANIK, MD, Peter J. 2131 Kenmore Road S Richmond, VA 23225	IM	AUTRY III, MD, Joseph Henry 11913 Geinsborough Road Palomac, MD 20854	P	DIAMANT, MD, Hermann 3100 Palm Aire Dr, Apt 505 Pompano Beach, FL 33060	IM
GROVES, MD, Cecil Dwight 5617 Pontiac Road Virginia Beach, VA 23462	OBG	MURPHY, MD, Maurice O. 107 Blake Road Norfolk, VA 23505	EM	STEIGMAN, MD, Cermen Kay 3338 Webley Court Annandale, VA 22003	PTH	BAGBY, MD, Richard Albert 908 Golfview Tampa FL 33609	OTO	DIETRICK, MD, Ronel Burton Presby Mission Box 213 Kwangju Korea 00100	GS
HA, MD, Chang Young 6810 Amster Road Richmond, VA 23225	PM	MYERS, MD, Russell H. 102 Burnham Place Newport News, VA 23606	FP	STIBAL, MD, Darlene M. 797 West 53rd St Norfolk, VA 23508	GS	BAILEY JR., MD, Wm Otis 2737 Devonshire Pl, Nw Washington DC 20008	IM	DOE, MD, Frederick David 500 West Alameda Roswell NM 88201	PTH
HADLEY, MD, Holly Weaver 380 Maple Avenue, West Vienna, VA 22180	FP	NEWTON, MD, C. Mark 507-I N Hamilton St Richmond, VA 23221	CD	STILL, MD, Gregory L. 11737 Jefferson Ave, Apt 10C Newport News, VA 23606	FP	BARNETT, MD, Charles P 7 Ivy Court Easton, PA 18042	PTH	DONOHUE, MD, Joyce C 1515 6th Avenue Birmingham, AL 35233	R
HALL JR, MD, James Allen 1547 Skirmish Run Drive Richmond, VA 23228	N	OLD, MD, Wayne David 201 Shirley Ave Norfolk, VA 23517	IM	STRAIN, MD, Brian M. 2404 Longview Ave, Apt 1-E Roanoke, VA 24014	GS	BEATY, MD, James Robt Rt 2, Box 11 Mayetta, KS 66509	EM	DRATLER III, MD, Siegfried 3800 Hillcrest Dr Apt 402 Hollywood FL 33021	A
HARCUS JR, MD, Sinclair J. 207 Greentree Park Charlottesville, VA 22901	FP	PANCOAST, MD, Sharon A. 806 West 49th Street Richmond, VA 23225	IM	TELL, MD, Alan M. 5715 Finborough Ct Richmond, VA 23228	OTO	BENDA, MD, Rudolf Anton Josef Austinville, VA 24312	GP	DUNCAN, MD, Victor Alberto P O Box 282 Huntsville, AL 35801	GS
HARR, MD, George C. 2401 Bennington Road Charlottesville, VA 22901	D	PARSONS, MD, Jerome M. Box 365, Mv Station Richmond, VA 23298	D	TEPORDE, MD, Gereldine M. 1016 South Wayne St, #707 Arlington, VA 22204	OM	BERNARDEZ, MD, Oscar Flores 56 Chelsea Way Road Bridgewater, NJ 08807	GS	DUNNE, MD, James Jos 3676 South Elm Lane Lentene FL 33462	PH
HARRIS, MD, Charles L. 3103 Carney Street Portsmouth, VA 23703	FP	PATEL, MD, Chandrakant M. Eastern State Hospital Williamsburg, VA 23185	*	TOMEU, MD, Enrique J. 1524 Hedgefield Ln Virginia Beach, VA 23456	OBG	BERRY, MD, Wm J 3737 Doctors Drive Port Arthur TX 77642	OBG	DUTT, MD, Cyril Prebheker 1434 Bethaven Road Riverdale, GA 30296	GS
HARRIS, MD, Mary Lawrence 1800 Jefferson Park Ave, Apt H Charlottesville, VA 22903	IM	PAYNE, MD, William R. Route 1, Box 862-A Barboursville, VA 22923	PD	TORRISI, MD, John Russell 2001 N Adams Street Arlington, VA 22201	TR	BLAKE, MD, Lynn French 301 Triplett Lane Knoxville TN 37922	PTH	ECHOLS, MD, Ches Little 350 West Thomas Rd Phoenix AZ 85013	N
HERSHEY, MD, J. Henry 615 Raleigh Avenue Norfolk, VA 23507	FP	PEARMAN, MD, Steven D. 1120 First Colonial Rd Virginia Beach, VA 23454	FP	TRIVETTE, MD, George A. 214 Whisperwood Glen Ln Reston, VA 22091	ON	BOGDAN, MD, Donald Fred 207 S Bedford St Georgetown, DE 19947	P	EDIS, MD, Theodore E 1108 Marine Wey West, Apt B-3L North Palm Beach FL 33408	P
HILL JR, MD, Lawrence K. 2000 N Adams St, Apt 330 Arlington, VA 22201	U	PIROS, MD, George P. Rr 3, Box 167 Charlottesville, VA 22901	DR	VAN WOLKENTEN JR, MD, Raymond 1446 Mallory Court Norfolk, VA 23507	FP	BOWYER, MD, John Victor 23165 Heiss Woodland Hills CA 91364	IM	EINSTEIN, MD, Norman Z 401 Mulberry St, Sw, #210 Lenoir, NC 28645	IM
HOFFMAN, MD, Michael J. 2206 Mandalay Drive, Apt E Richmond, VA 23224	GS	POLIQUIN, MD, James R. P O Box 843 Norfolk, VA 23501	GS	VANDEBERG, MD, Byron F. 1336 Blackrock Drive Richmond, VA 23225	CD	BRITTINGHAM JR., MD, L A P.O. Box 2613 Everett, WA 98203	DR	ELLIS, MD, Herman Martin 744-23 Copper Rd Geithersburg, MD 20878	GPM
HOGAN, MD, Elizabeth W. 2806 Woodlawn Ave. Falls Church, VA 22042	FP	POLLART, MD, Susan M. 1516 Broad Ave Charlottesville, VA 22903	FP	VIEWEG, MD, W.V.R. 1715 Kenwood Lane Charlottesville, VA 22901	P	BROWER, MD, Anne Clayton 3800 Reservoir Rd Washington, DC 20007	DR	ERRICO, MD, James Melton 100 Westwood Avenue High Point NC 27262	OPH
HOLT JR, MD, Frank Loving 417 S Fillmore St Arlington, VA 22204	GE	QUINONES, MD, Moises E. 4201-A Yellow Mtn Rd Roanoke, VA 24014	U	VORSTMAN, MD, Albert W. 5559 Old Guerd Crescent Virginia Beach, VA 23462	U	BROWN, MD, Lee Buckingham 926 E Mc Dowell Rd Phoenix AZ 85006	TS	FABIE, MD, Anastacie E. 514 Pine Street Cebol, MO 65889	IM
HOROWITZ, MD, Jed H. 1722 Verma Pl Charlottesville, VA 22901	PS	RAY, MD, Brenda J. 6717-B Crenshaw Rd. Richmond, VA 23227	IM	WATERS, MD, Barry K. 7353 Longview Drive Richmond, VA 23225	RHU	BRUMFIELD JR., MD, Robert H 1808 Verdugo Blvd Glendale CA 91208	ORS	FALK, MD, Leo J. 5315 Strehmore Ave Kensington, MD 20895	IM
IRBY, MD, Jane Craig 5803 Westover Drive Richmond, VA 23225	IM	REINHARDT, MD, Stephen D. 4405 Forest Hill Ave Richmond, VA 23225	FP	WATERS, MD, Susan W. 7353 Longview Drive Richmond, VA 23225	PD	BRYAN, MD, Paul A 111 W Montgomery Ave Rockville MD 20850	GP	FARROW, MD, Creston 3000-A Walnut Grove Rd Memphis TN 38111	PTH
JACKSON, MD, Leroy T. 6420 King Louis Dr Alexandria, VA 22312	IM	RENALDO, MD, Gary Joseph 688 Elgin Terrace Richmond, VA 23225	IM	WEISSBERGER, MD, Marshall A. 10037-A Palace Court Richmond, VA 23233	IM	BUTTERY, MD, Christopher M G P O Drawer 9727 Corpus Christi, TX 78408	FP	FELDMAN, MD, Daniel M. 2000 N Petterson St Veldoste, GA 31602	PD
JOYCE, MD, Judith Marie Route 9, Box 149 Charlottesville, VA 22901	R	RHEA, MD, Randall R. 2213 Stanley Ave, Sw, #6 Roanoke, VA 24014	FP	WIESE, MD, Kurt L. 769 E Mountain Wood Rd Charlottesville, VA 22901	ID	BYERLY, MD, Baxter H 1330 Micosukee Rd, #201 Tallahassee FL 32303	OPH	FLEIGEL, MD, Jeffrey Dee 1224 S Magnolia Ext Ocala, FL 32670	OTO
KALTREIDER, MD, Sara A. 5206 King William Rd Richmond, VA 23225	OPH	RICE, MD, Diane Beach 3300 Gallows Road Falls Church, VA 22046	PTH	WINELAND, MD, Richard H. 225 Coffee Rd, #38 Lynchburg, VA 24503	FP	BYRD JR, MD, Walter R. 2116 Arapaho Road E, #581 Richardson, TX 75081	P	FREDA, MD, Franklin D 8775 20th St, Lot 281 Vero Beach, FL 32960	PD
KENNEDY, MD, Mary M. 601 Pembroke Ave, Apt 401 Norfolk, VA 23507	PD	RIGGLE, MD, Karl P. 321 East Jerald Street Highland Springs, VA 23075	GS	WYATT, MD, David A. 1610 Greenleaf Lane Charlottesville, VA 22903	U	CALA, MD, Benjamin B 2561 Sesame Court Spring Hill, FL 33526	U	FREED, MD, Thos Alexander Merin Gen Hosp Radiology Dept Greenbree CA 94902	DR
KERSH, MD, Charles R. 513 Overcliff Court Midlothian, VA 23113	TR	ROBERTS II, MD, Allen H. 1356 Llewellyn Ave Norfolk, VA 23517	IM	YANESSA, MD, Richard J. 9001 Cloisters East Richmond, VA 23229	PMH	CALZADILLA, MD, Miguel R 5881 Sw, 7th Street Plantation, FL 33317	PD	FUCHS III, MD, Kaspar 1526 North Edgemont St Los Angeles CA 90027	NS
KOLLER, MD, Miriam K. 104 W Hillcrest Ave Richmond, VA 23226	CHP	ROSENTHAL, MD, James 910 Colonial Ave Norfolk, VA 23507	FP	YOUNGER, MD, Deborah A. Route 1, Box 98 Palmyra, VA 22963	CHP	CARROLL, MD, Robert P. 4942 Ne Stalling Dr Nacogdoches TX 75961	FP	FUNK, MD, Richerd Ludwig 6050 Offenbach/Mein Beltline Strasse 13 Germany West G0407	PTH
KUBAN, MD, Deborah A. 711 Westover Ave Norfolk, VA 23507	ON	ROTHMAN, MD, Alan Lee 1330 Greenmoor Drive Richmond, VA 23225	IM	ZACHOW, MD, Steven E. 3343 Warner Road Richmond, VA 23225	R	CARTER, MD, Edward Kent 2200 Pendragon Rd Kingsport TN 37660	R	GABRIEL, MD, Daniel E. 114 Woods Lane Mc Murrey, PA 15317	IM
KUNDRAT, MD, Andrew G. 150 Mill Point Drive Hampton, VA 23669	IM	SADOVE, MD, Richard C. 400 W Brambleton Ave, #300 Norfolk, VA 23510	PS	ZAVOSKI, MD, Robert Wood 728 W Prin Anne Rd, Apt 1 Norfolk, VA 23517	PD	CAVEDO JR, MD, Irvin Walters 127 Ocean Blvd W Supply NC 28462	DR	GALLAGHER, MD, M. E. 400 Young St Melbourne FL 32935	OM
LAUDONE, MD, Vincent P. 114 Denice Lane Charlottesville, VA 22901	U	SAKOW, MD, Nolan K. 330 W Brambleton Ave, #1812 Norfolk, VA 23510	PTH	OUT OF STATE					DR
LEVINE, MD, Richard A. 2208 S. Jefferson St Roanoke, VA 24014	IM	SANDERSON, MD, Timothy A. 841 Redgate Avenue Norfolk, VA 23507	P	ABBOTT, MD, Geo Richard 153 Hollyvale Dr Rochester NY 14618	PTH	CHURCH, MD, David Russell 137 Monte Rey Los Alamos, NM 87544	GP	GATES JR., MD, Herbert S P O Box 707070 Tulsa, OK 74170	OBG
LEVITT, MD, Lynn C. 2808 Sweetbriar Ave, Sw Roanoke, VA 24015	FP	SCOTTINO, MD, Mary Anne 150 Mill Point Drive Hampton, VA 23669	FP	ADEVOSO, MD, Lauro L. P O Box 16206 Rochester, N.Y 14616	AN	COLEMAN, MD, Wm P 4330 Loveland St Metairie LA 70002	A	GERGEN, MD, Werner Anthony Guthrie Clinic Seyre PA 18840	OBG
LLEWELLYN, MD, Christine H. 3809 Gill Street Chester, VA 23831	DR	SENATORE, MD, Peter J. 1415 North Oak Street Arlington, VA 22209	GS	ALLENDE, MD, Jorge A Po Box 277 Shady Spring WV 25918	OPH	COLLISON, MD, Edward Frank Wilson Memorial Hospital-Xray Wilson, N.C 27893	DR	GHAREMANI, MD, Gery 800 Sheridan Road Highland Park, IL 60035	DR
LONGERAN, MD, Maryrose T. 4400 North 4th Road, #2 Arlington, VA 22203	AN	SHAH, MD, Mukesh H. 12970 A 7 Nettles Dr Newport News, VA 23602	P	ALPERN, MD, Frederick P 103 Windblown Court Baltimore, MD 21209	PD	COSTA, MD, Giovanni Giacomo 6300 Powers Road Orchard Park, NY 14127	*	GILLESPIE, MD, Frederick D 1205 Market St Parkersburg WV 26103	OPH
LOUKA, MD, Kamal S. 500 Pacific South, #1008 Virginia Beach, VA 23451	FP	SIGAL, MD, Barry W. 508 Rossmore Road Richmond, VA 23225	IM	ALVIG, MD, Olav Henry Forsyth Co Hosp, Box 768 Cumming, GA 30130	DR	CRAWFORD, MD, John Custis Box 293 Nags Head NC 27959	OBG	GORMLEY, MD, Robt Arthur 10302 Florian Road Louisville, KY 40223	IM

\* = AMA membership



# EATING DISORDERS

## DOMINION HOSPITAL

**is pleased to announce the opening of its  
treatment program for Eating Disorders**

The Dominion program has utilized the latest in psychodynamic and biological approaches to design a unique program exclusively for the diagnosis and treatment of anorexia nervosa and bulimia. Drawing on the expertise of two nationally recognized experts on Eating Disorders, the program is intended to make long-term changes in the eating behaviors of anorectics and bulimics and restore the ability to eat normally without obsessive concerns about weight and food.

Medical monitoring and individually designed treatment programs are special features of the program which also offers:

- Individual, group, family, and multifamily therapy
- Nutritional education and therapy, including cooking classes
- Life skills, assertiveness training, art therapy, exercise program, and leisure skills
- Therapeutic milieu therapy
- Pharmacotherapy

Treatment of anorectics will focus on weight restoration and weight maintenance, while treatment for bulimics will focus on establishing stable meal patterns before discharge from the hospital.

Dominion Hospital is a fully accredited private psychiatric hospital offering adolescent and adult psychiatric treatment, chemical dependency treatment, and The Dominion School.

### 24 HOUR ADMISSIONS

Admissions and Information

**703/536-2000**

### Dominion Hospital

2960 Sleepy Hollow Road, Falls Church, Virginia 22044

## 36 OUT OF STATE

GRAHAM JR., MD, John Calhoun 106 S. Water Street P.O. Box 250 Elizabeth City NC 27909	DR	INGRAM, MD, Phyllis Ray 50 Dogwood Court Daniels WV 25832	GS	MAC PHERSON, MD, Archibald R 331 N Maitland Ave Maitland FL 32751	PD	POWELL, MD, John David 328 Robin Rd Mount Airy NC 27030	GS	TABOADA, MD, Alberto De Jesus 3 Twilight Drive Brick, NJ 08723	AN
GRAHAM, MD, Cecil Cleveland Route 2, Box G-20 Hammond, LA 70401	AN	JANSEN, MD, George Allen 1042 Rota Dr - Aalt Apo/SIO 96334	AN	MACON, MD, Edward Malcolm 112 Hartle Drive Hagerstown, MD 21740	EM	PROPPER, MD, Norman S 102 East Ravind Rd Kingsport TN 37660	OBG	TABOADA, MD, Juanito Corrales 582 Se 7th Avenue Crystal River, FL 32629	GP
GRAY, MD, Edwin H Univ Texas Box 7339 Austin TX 78712	P	JENNINGS, MD, Thornton S 517 South Mildred Charles Town, WV 25414	GP	MAFFEY, MD, Ralph B 189 Midland Ave, #3 Kearny NJ 07032	FP	RAVITZ, MD, Gerald Alan Rd #1 Box 130R Schuylkill Haven, PA 17972	U	TAM, MD, Thomas L H 407 Arbour Drive Savoy, IL 61874	DR
GRAY, MD, Mary C. P.O. Box 2138 Jacksonville, TX 75766	CHP	JOHNS, MD, Michael Edward 600 N Wolte St, 466 Carnegie Baltimore, MD 21205	OTO	MAGEE, MD, Archibald C. Route 3, Box 561 Washington, NC 27889	*	REDFORD, MD, John W B Rainbow Blvd & 39th St Kansas City KS 66103	PM	TAN, MD, Jose G 2890 Greenbrooke Drive Olmsted Township, OH 44138	FP
GRIFFIN JR., MD, Edward E 931 East 86th Street Indianapolis, IN 46240	ORS	JOHNSON, MD, L Meredith 213 Richmond Hill Drive Asheville, NC 28806	PUD	MALDONADO, MD, Luis Gonzalo Signal Mountain, TN 37377	DR	RELYEA, MD, Richard Lee 3112 Connors Drive Las Vegas NV 89107	OPH	TEXTER JR., MD, John Henry Box 3926, Div 01 Urology Springfield, IL 62708	U
GRISSBY JR., MD, Wm C 249 Midway Street Bristol, TN 37620	OBG	JOHNSON, MD, William Waldo 1207 Fairchild Court Woodland, CA 95695	OTO	MALEK, MD, Nabil Shehate Rizk 29025 Bolingbrook Mayfield Heights OH 44124	AN	RHODE, MD, Joseph G. 1020-B Andrews Hwy Midland, TX 79701	FP	THOMAS, MD, Gordon Clark G Rd #3 Daisy Gardner Rd Laconia NH 03246	P
GROUSE, MD, Lawrence Douglas 1200 Brooks Lake Downers Grove Ill 60515	IM	KABLE, MD, Kelvin D. P.O. Box 1098 Concord, NC 28025	AM	MALONEY, MD, Wm F 5901 E 7th Street Long Beach, CA 90822	IM	RICHARDS, MD, A Dewey 303 Main Street Orono, ME 04473	FP	THORNHILL, MD, Wm Rathborn 902 Hathwa Drive Anderson, SC 29621	GP
GRYTE, MD, Clifford F P.O. Box 822 Huron SD 57350	IM	KASINOFF, MD, Bernard H P.O. Box 917 Lebanon PA 17042	P	MANN, MD, Geoffrey T 227 Codrington Drive Fort Lauderdale FL 33308	PTH	RICHTER, MD, Paul 2739 Felton Dr, #110 East Point GA 30344	NEP	TOFFOLO, MD, R. Ronald 1205 Wales Road Kings Mountain, NC 28086	R
GWINN, MD, James Alexis Box 598 Lockbridge, WV 25973	EM	KAUFFMAN JR., MD, Herbert M 9200 West Wisconsin Ave Milwaukee WI 53226	GS	MARTIN, MD, Carolyn Jean 707 Stone Lake Cieburne, TX 76031	PTH	RIEMAN, MD, G Fletcher 2148 Echo Lane Wilmington NC 28403	OBG	TUSING, MD, Thos Wm 13561 Featherstone Dr St Louis MO 63131	OS
HALEY, MD, Harold Bernard V.A. Medical Center 2002 Holcombe Blvd Houston, TX 77211	GS	KEARNEY JR., MD, Rochoert W 1408 Madison Avenue Mankato, MN 56001	ORS	MC COLLUM, MD, Donald C 8 Brushwood Rd Asheville NC 28804	PH	RIZKALLA, MD, Sam N 453 77th St Brooklyn NY 11209	U	TUTAK, MD, Unal 2100 16th Ave, S, # 205 Birmingham AL 35255	AN
HALL, MD, Robt Walker Post Office 1117 Arcadia, FL 33821	R	KENNAN JR., MD, Richard Barnes 526 West College Avenue Salisbury, MD 21801	P	MC DANIEL JR., MD, Eugene M 1142 North Road Street Elizabeth City, NC 27909	OBG	ROBERT, MD, Dyer 1007 N 6th Street Albemarle NC 28001	U	TYLER, MD, John Hutchinson 5850 Calder, Apt 220 Beaumont, TX 77706	GP
HAMLETT, MD, Luther J P.O. Box 8002 Sisters, OR 95579	OBG	KENYON JR., MD, Robert Earl Po Box 1418 Wallington, CT 06492	IM	MC FADDIN, MD, James G 28 Midway St Bristol TN 37620	ORS	ROSENBAUM, MD, Geo R Route 2, Box 17 Denver, CO 28037	OST	VADEEN, MD, Edwin Booth The Oystercatcher Pawleys Island, SC 29585	PD
HAMPshire, MD, Alan Curtis Alexandria V A Hospital Alexandria LA 71301	P	KERSEY JR., MD, Wm Ward Peterstown WV 24963	GP	MC GEE JR., MD, James E 1700 Brand Street Bluefield WV 24701	GS	ROSENBERG, MD, Leon Harris 245 22 Street, Nw Canton, OH 44709	PTH	VAN DEN BRANDEN, MD, Frederik M P 10044 South A.1.A., Apt #602 Jensen Beach, FL 33457	M P
HAMPTON, MD, Archibald S Rt 2, Box 142 Alachua FL 32615	PTH	KREUTZ, MD, Berny J 9715 Med Cen Dr, #435 Rockville, MD 20850	AN	MC RAE, MD, Marvin E 344 N Elm St Greensboro NC 27401	D	ROSENBERG, MD, David Irving P.O. Box 462 Flemington NJ 08822	U	VAN REKEN, MD, David E. 6030 W Roosevelt Road Oak Park, IL 60304	PD
HANSBARGER, MD, Echols Alcott P.O. Box 2548 Charleston WV 25329	PTH	KINDRED, MD, Robert G 5411 W Cedar Ln Bethesda MD 20014	ORS	MELTON III, MD, John Wesley 1234 - 19th St Nw Washington DC 20036	RHU	ROSENBERG, MD, David Irving P.O. Box 462 Flemington NJ 08822	U	VARNER, MD, Donald Wayne 346 Honeycutt Drive Wilmington, NC 28403	PTH
HANSON, MD, Maury Lloyd 2-G Booker Ck Apts Chapel Hill, NC 27514	NS	KIRKPATRICK, MD, Saml A 249 Alleghany Ave Hanover PA 17331	OBG	MITCHELL, MD, Robt Hartwell 15 Ocean Dr Punta Gorda FL 33950	OPH	ROSSILLO, MD, Ludwig Anthony 4322 Bream Road Charleston Hts, SC 29418	GS	VAZQUEZ, MD, Hariberto 205 N. Sequoia Drive West Palm Beach, FL 33409	P
HARKRADER JR., MD, Charles J 21 Compton Road Middlebrook Bristol TN 37620	GS	KREUTZ, MD, Berny J 9715 Med Cen Dr, #435 Rockville, MD 20850	GS	MITSOPoulos, MD, Peter M 290-174 Street Apartment 518 Miami Beach, FL 33160	IM	RUWIVAR, MD, Felix Santos 1521 N Wilcox #210 Hollywood, CA 90028	R	VELASQUEZ, MD, Emilio T. One Edgewood Court Chillicothe, OH 45601	P
HARRIS, MD, Robt Eldred 4499 Med Dr, #171 San Antonio TX 78229	OBG	LAMB, MD, Wm Robt Torch Box C Traverse City, MI 49684	CHP	MOORE, MD, Peter V 4901 Hillbrook Ln Nw Washington DC 20016	CHP	SANDERS, MD, Margaret Mackie 434 Fiedwood Drive Richardson, TX 75081	U	VITOLS, MD, Edith T. 1809 Evergreen Ave Goldsboro, NC 27530	US
HARTER, MD, Basil T 249 Midway St Bristol TN 37620	OBG	LANGDALE, MD, Emory L 1064 Stonehenge Dr Hanahan, SC 29406	PM	MORGAN II, MD, Rees 5100 N Ocean Blvd 417 Fort Lauderdale FL 33308	OBG	SCHURTER, MD, Lonis Leon 505 Northwood Circle Garner NC 27529	GS	VON ARX III, MD, Emil K Dept Of State Washington DC 20520	GS
HELENBOLT, MD, Kenneth S 3563 Longhollow Rd Fargo ND 58102	U	LARSON, MD, Lawrence Oliver 1421 Crawford Ln Ann Arbor, Michigan 48105	GS	MORGENSTERN, MD, Jack Arnold P.O. Box 7067 Macon, GA 31210	P	SCIALLI, MD, John V. K. 5051 North 34th St Phoenix, AZ 85018	PTH	WAGNER, MD, Wm Framont 201 Lee Avenue Highland Springs, VA 23075	GS
HEMMERLEIN, MD, Arthur H 111 Arthur Avenue Clarksburg, WV 26301	FP	LAUDE, MD, Walter Edwin H 444 Seminole Road Babson Park FL 33827	AN	MORRISON, MD, Robt Lord Route #4 - Box 433A Wilmington NC 28405	U	SEIGER, MD, Julian Wood 1210 Riverside Avenue Elizabeth City, NC 27909	GS	WATERS JR., MD, John A 15101 Sw 87th Ave Miami, FL 33176	OPH
HERGENROEDER, MD, Paul J P.O. Box 63 Richfield, OH 44286	OBG	LAUPUS, MD, Wm Edward 218 Country Club Dr Greenville NC 27834	PD	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	AN	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	P	WELLS, MD, Chas Lewis Box 2000 Fayetteville NC 28302	TS
HESS, MD, Leonard Wayne 10948 Middleboro Drive Damascus, MD 20872	OBG	LAWSON, MD, William J. P.O. Box 338, Va Med Cen Dayton, OH 45428	AM	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	P	WELLS, MD, Lewis Emmor 3919 Se 9th Court Cape Coral FL 33904	IM
HIMES, MD, Thomas R 319 Walnut St Roverstord, PA 19468	PTH	LAWTON, MD, George Marion P.O. Box 808 (L423) Livermore, CA 94550	OM	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH	WETZLER, MD, Richard Austin 4568 Wagon Wheel Dr Birmingham MI 48010	GP
HITREC, MD, William S. 27852 Puerta Real Suite 110 Mission Viejo, CA 92691	GP	LEA, MD, Jos Davis Alburtuck Farm Port Harbor NC 27964	IM	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH	WHELAN, MD, John Jos 58 Springfield Rd Box 565 Somers CT 06071	P
HOCH-LEGITI, MD, Cornelia P.O. Box 340 Shepherdstown WV 25443	GS	LEAVENS, MD, Deborah I. Bad Kessingen Health Clinic Apo, New York 90330	FP	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH	WILLIAMS, MD, John Stuart Rt 1, Box 181 Hilliard, FL 32046	EM
HODGES, MD, Thos O V.A. Medical Center Biloxi MS 39531	IM	LEE JR., MD, James Harold 1025 Walnut St Philadelphia PA 19107	OBG	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH	WINSTEAD, MD, Glenn C. 4801 Pepperidge Odessa, TX 77611	U
HOENE, MD, Rudolt W Rtd #2 Durham NH 03824	R	LEONE, MD, Louis August 593 Eddy St Rhode Island Hosp Providence RI 02903	ND	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH	WOODWARD JR., MD, Edward 858 Errol Parkway Apopka FL 32703	IM
HOLT, MD, Bevely Dan 312 North Main St Greenville, TN 37743	ORS	LEONHARDT, MD, Hannah V 108 Holbrook Ave Danville, VA 24541	PTH	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH	WYRE, MD, John C P.O. Box 1267 Morro Bay CA 93442	P
HOOPER, MD, Roy Michael 3118-B Middlebrooks Cir Tallahassee FL 32303	AN	LEONHARDT, MD, Hannah V 108 Holbrook Ave Danville, VA 24541	PTH	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH	WU, MD, Hsin Hsiung 45621 Harmony La Belleville MI 48111	AN
HORNE, MD, Francis G Rtd 1, Box 320 Fort Spring, WV 24936	AN	LOAR, MD, Charles Richard 3803 Emerson Ave Parkersburg W Va 26107	NS	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH	YAGER, MD, John Julius Box 2026 Sta A White Sulphur Spgs WV 24986	DR
HORNE, MD, Melvin L 2600 Sandfiddler Road Virginia Beach, VA 23456	IM	LOUW, MD, Jan Cornelius 16 Argyle St, Riepen Hall Sandton, Rep South Afric 02196	P	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH		
HOWELL JR., MD, Edgar V P.O. Box 1148 Rockingham, NC 28379	ORS	LUCARIC JR., MD, Zdenko 17640 Osborne Northridge CA 91324	IM	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH		
HUMPHRIES, MD, Wm C 13 Tanglewood Rd Black Bks St Simons Island GA 31522	GS	LUCAS, MD, Thos L 31 State St Charleston SC 29401	FP	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH		
HUNDLEY, MD, Jos Leigh 320 N Magnolia Ave Orlando FL 32801	D	MAC KNIGHT, MD, Jos Chas Du Pont Company Seaford, DE 19973	IM	MORRIS, MD, Frances C. 1935 Amelia Dallas, TX 75235	PD	SELTZER, MD, Geo Breaker 2115 Springdale Dr Newberry SC 29108	PTH		



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BICKERS, MD, Wm M	Richmond	BORGES, MD, Albert Facundo	Fairfax	BROOKS JR., MD, Geo K	Richmond	BURWELL, MD, Brownen	Southside Va
BIDWELL JR., MD, Glenn Porter	Tri-County	BORGES, MD, Philip M	Arlington	BROOKS JR., MD, John	Norfolk	BURWELL, MD, James Abraham	Fairfax
BIGELOW, MD, Llewellyn Barry	Alexandria	BORILLO, MD, Romeo Bisquera	Southwest Va	BROOKS, MD, Charles Harris	Rockingham	BURWELL, MD, Lawrence R	Albemarle
BIGLEY JR., MD, Elmer C	Alexandria	BORING, MD, Wayne Douglas	Norfolk	BROOKS, MD, James W	Richmond	BUSCHI, MD, Anthony J	Albemarle
BIGLEY JR., MD, H Alan	Southside Va	BORLAND, MD, David S	Williamsburg	BROOKS, MD, Kenneth Phillip	Richmond	BUTLER III, MD, William W.	Roanoke
BIGONEY, MD, Rebecca M	Fredericksburg	BORTNICK, MD, Ronald Jack	Fairfax	BROSHAN, MD, Kathleen Angela	Richmond	BUTLER JR., MD, Bruce	Alexandria
BINDER, MD, Monte Leroy	Newport News	BORUCHOW, MD, Lillibeth B.	Arlington	BROWER, MD, Anne Clayton	Out Of State	BUTLER, MD, Lilia G	Alexandria
BINDER, MD, Richard Allen	Fairfax	BOSHER JR., MD, Lewis Hinton	Richmond	BROWN III, MD, Alexander G	Richmond	BUTLER, MD, Thomas Parke	Arlington
BINFORD, MD, Chapman Hunter	Arlington	BOST, MD, Michael Anthony	Richmond	BROWN III, MD, Jos Danl	Williamsburg	BUTTERWORTH III, MD, John F	Richmond
BINFORD, MD, Charles A	Richmond	BOST, MD, J. Thornton	Orange	BROWN JR., MD, Leon Junius	Richmond	BUTTERWORTH, MD, Thomas R	Richmond
BINHAMMER, MD, Harold E	Richmond	BOSWELL, MD, David C	Fairfax	BROWN, MD, Cyrus U.	Hampton	BUTTERY, MD, Christopher M G	Out Of State
BINKS, MD, Silas O	Hampton	BOSWORTH, MD, David C	Newport News	BROWN, MD, Edwin Merriman	Richmond	BUTTS, MD, Edward B.	Virginia Beach
BIRDSONG, MD, Gordon G	Tri-County	BOSWORTH, MD, E. W.	Accomack	BROWN, MD, James Geo	Alexandria	BUTZNER JR., MD, Wm Walker	Fredericksburg
BIRO, MD, Victor G	Richmond	BOSWORTH, MD, James Elam	Virginia Beach	BROWN, MD, James La Velle	Richmond	BUXTON III, MD, Ernest Perry	Newport News
BISESE, MD, Albert Jos	Norfolk	BOTT, MD, D. Gregory	Northern Va	BROWN, MD, Kimbarley L.	Residents	BUXTON JR., MD, Ernest Perry	Richmond
BISHOP, MD, William D.	Southwest Va	BOTTON, MD, Jacques Ephraim	Lynchburg	BROWN, MD, Lee Buckingham	Out Of State	BUXTON, MD, Martin N	Richmond
BISHOP, MD, Wm Branch	Southside Va	BOULWARE, MD, Ralph H.	Lynchburg	BROWN, MD, Margaret Ann	Residents	BUXTON, MD, Russell V	Newport News
BIVENS JR., MD, Carl Hill	Roanoke	BOURGARD, MD, Lawrence Dean	Virginia Beach	BROWN, MD, Peter Wilcox	Fairfax	BUXTON, MD, William D.	Albemarle
BIVINS, MD, Don Howard	Roanoke	BOURGEAIS, MD, F. John	Albemarle	BROWN, MD, Raymond Sidney	Mid-Tidewater	BYER, MD, Barry	Fairfax
BLACK JR., MD, James B	Richmond	BOURNE, MD, Henry Raid	Danville	BROWN, MD, Robert Stanley	Albemarle	BYERLY, MD, Baxter H	Out Of State
BLACK, MD, Yuill	Arlington	BOUTROS, MD, Samir B.G.	Southside Va	BROWN, MD, Ronald Bayard	Norfolk	BYERS, MD, Francis L	Rockingham
BLACKBURN, MD, James E	Lynchburg	BOWDEN JR., MD, Robt Henry	Lynchburg	BROWN, MD, Thos Mc Pherson	Arlington	BYRD JR., MD, Walter R.	Out Of State
BLACKMAN, MD, Raymond S	Albemarle	BOWEN, MD, Courtney C	Southwest Va	BROWN, MD, Toby L.	Prince William	BYRD, MD, Chas Wm	Richmond
BLACKMON, MD, Wayne D	Alexandria	BOWEN, MD, Patrick J	Tazewell	BROWN, MD, William Martin	Lynchburg	BYRD, MD, John Abbott	Norfolk
BLACKMORE, MD, John Robt	Richmond	BOWEN, MD, Robert R	Fairfax	BROWN, MD, William P.	Chesapeake	BYRD, MD, Wm Edward	Norfolk
BLACKWELL, MD, James Edward	Roanoke	BOWER, MD, Richard Edward	Lynchburg	BROWNE, MD, Roger Wayne	Halifax	BYRNE, MD, Robert Eugene	Arlington
BLADES, MD, James Franklin	Richmond	BOWERS, MD, John T.	Tazewell	BROWNLEY, MD, Edwin T.	Norfolk	BYRNE, MD, Robert Francis	Virginia Beach
BLAIR III, MD, Chas Jos	Richmond	BOWLES, MD, Russell V	Norfolk	BROWNLEY, MD, Harvey C	Lynchburg	BYRNE, MD, Wm Draper	Fairfax
BLAIR, MD, Walter Bernard	Roanoke	BOWLES JR., MD, Richard B	Fairfax	BROWNSTEIN, MD, Willis Edwin	Fairfax		
BLAIR, MD, Wm F	Norfolk	BOWLES, MD, James H	James River	BRUBAKER, MD, Herman W	Roanoke		
BLAKE, MD, Jeffrey D.	Alexandria	BOWLES, MD, James William	Southside Va	BRUCE JR., MD, James Garnett	Orange		
BLAKE, MD, Lynn French	Out Of State	BOWLES, MD, Paul Elwood	Roanoke	BRUCH, MD, Wm Mark	Richmond		
BLAKE, MD, Michael Clarence	Southside Va	BOWLES, MD, Richard Boxley	Mid-Tidewater	BRUCKNER, MD, Jana Tarezie	Virginia Beach		
BLAKE, MD, William	Fairfax	BOWMAN, MD, John David	Richmond	BRUCKNER, MD, Nancy V	Fairfax		
BLAKELY, MD, Lee Add	Fairfax	BOWSER, MD, Barrington H	Richmond	BRUGH III, MD, Victor Miller	Richmond		
BLAKEY, MD, Hubert H	Alexandria	BOWYER, MD, John Victor	Out Of State	BRUMFIELD JR., MD, Robert H	Out Of State		
BLAKEY, MD, Peter P.	Richmond	BOYAN, MD, Chas P	Richmond	BRUMMER, MD, Donald Louis	Richmond		
BLALOCK, MD, Jos Rogers	Southwest Va	BOYCE JR., MD, Edward L	Richmond	BRUNK, MD, James Robt	Rockingham		
BLANCHARD III, MD, Lawrence E	Richmond	BOYCE, MD, Stanley Carlton	Richmond	BRUNO JR., MD, Alphonse H L	Chesapeake		
BLANCHARD, MD, P.B	Virginia Beach	BOYD JR., MD, John Otto	Roanoke	BRUNO JR., MD, John A	Alexandria		
BLAND, MD, David L	Hampton	BOYD, MD, James Horace	Fairfax	BRUNO, MD, Peter D.	Fairfax		
BLANK, MD, Alvin Robert	Virginia Beach	BOYD, MD, Milton Alexander	Richmond	BRUSH JR., MD, Edward V	Rockbridge		
BLANKENBAKER, MD, Walter L	Albemarle	BOYD, MD, Robt Stewart	Northern Va	BRUSH, MD, John J.	Richmond		
BLANKINSHIP, MD, Rex	Richmond	BOYD, MD, Wm Everett	Norfolk	BRUST, MD, Stuart Wm	Lynchburg		
BLANTON JR., MD, Wyndham B	Richmond	BOYER, MD, A Stephen	Northampton	BRUTHER, MD, Lawrence James	Fairfax		
BLANTON JR., MD, Frank	Southwest Va	BOYER, MD, Paul Henry	Fairfax	BRYAN JR., MD, Phillips R.	Augusta		
BLANTON, MD, Erika M	Richmond	BOYS, MD, Floyd Eugene	Albemarle	BRYAN, MD, Louis	Arlington		
BLANTON, MD, Frank M	Richmond	BRADENHAM, MD, Ben Persons	Richmond	BRYAN, MD, Paul A	Out Of State		
BLASCO, MD, Peter A	Albemarle	BRADLEY, MD, Chester Dale	Hampton	BRYAN, MD, Phillips	Lynchburg		
BLATTNER, MD, Carlos Luis	Southwest Va	BRADLEY, MD, Robert W	Richmond	BRYANT JR., MD, John E	Newport News		
BLAY, MD, Andrew Brian	Rockingham	BRADSHAW, MD, Douglas M	Residents	BRYANT JR., MD, Carol A	Tri-County		
BLAYLOCK, MD, Wilmer Kenneth	Richmond	BRADY JR., MD, John W	Prince William	BRYANT, MD, Robt Singleton	Albemarle		
BLAYLOCK, MD, Wm Mc Gehee	Roanoke	BRAGG, MD, Leroy P	Hampton	BRYANT, MD, Stephen Robert	Rockingham		
BLAYTON, MD, James Blaine	Williamsburg	BRAM, MD, Frederick Martin	Fairfax	BRYCE II, MD, Edwin Clinton	Lynchburg		
BLEI, MD, C. Lynne	Arlington	BRAMMER, MD, Paul William	Fredericksburg	BRYCE JR., MD, Wm Fielding	Richmond		
BLEY, MD, Donald E	Fredericksburg	BRANCH, MD, David W	Roanoke	BRYSAN, MD, Gilbert Hamilton	Richmond		
BLISS III, MD, Theodore	Norfolk	BRAND, MD, Eugene D	Northern Neck	BUCHANAN, MD, Brian D.	Bedford		
BLISS, MD, Reba N Gwyneth	Fairfax	BRAND, MD, Rolf	Southside Va	BUCHANAN, MD, Chas Stuart	Northern Va		
BLITCH JR., MD, James Bedford	Fairfax	BRANDT, MD, Kurt T.	Fairfax	BUCHANAN, MD, John Goodwin	Portsmouth		
BLIZZARD, MD, Robt M	Albemarle	BRANDT, MD, V.	Fairfax	BUCHANAN, MD, Robt James	Chesapeake		
BLOCK, MD, David A	Fairfax	BRANN, MD, Wm Cralle	Halifax	BUCHER, MD, Bruce M.	Mid-Tidewater		
BLOCK, MD, Leon Irving	Fairfax	BRANSCOME, MD, William C	Augusta	BUCK JR., MD, Frank Neville	Lynchburg		
BLOOM, MD, Marvin Eugene	Fairfax	BRANSON, MD, Donald Gene	Lynchburg	BUCUR, MD, John C	Arlington		
BLOOM, MD, Robert L	Fairfax	BRANTLEY JR., MD, Julian	Fairfax	BUDD JR., MD, Saml Walthall	Richmond		
BLOSE, MD, Donald Curtis	Southwest Va	BRANTLEY, MD, Aurelius Walter	Southside Va	BUENAVENTURA, MD, Francisco	Alexandria		
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BOARD, MD, Anne J Woodrum	Richmond	BRAY, MD, Maurice Miller	Tri-County	BULLOCK JR., MD, Henry A	Richmond		
BOARD, MD, John Arnold	Richmond	BRAY, MD, Stuart Thomas	Albemarle	BULLOCK JR., MD, John W	Richmond		
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BOATWRIGHT, MD, Chas Lee	Southwest Va	BREDRUP JR., MD, Ole C	Richmond	BULLOCK, MD, Robt Graham	Arlington		
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BODNER, MD, Bruce Ira	Norfolk	BRENNBRIDGE, MD, A Norman A G	Albemarle	BUNDY JR., MD, Walter E	Richmond		
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BOGGESS, MD, H. Preston	Roanoke	BRESSLER, MD, Bernard	Norfolk	BURCH, MD, John Gordon	Williamsburg		
BOHAN, MD, Michael E	Portsmouth	BREWER, MD, Herbert Martin	Albemarle	BURDICK, MD, Edward Peter	Williamsburg		
BOHLKE, MD, Glen Leroy	Virginia Beach	BREWER, MD, Richard James	Virginia Beach	BURGE, MD, Joseph John	Albemarle		
BOKINSKY, MD, Gary Brooks	Richmond	BREWER, MD, Robt Geo	Richmond	BURGER JR., MD, Ray Edward	Northampton		
BOLAND, MD, Brian J	Arlington	BREWER, MD, William Henry	Residents	BURGER, MD, Robt Lindsay	Norfolk		
BOLEN, MD, John Wm	Southwest Va	BRICKFIELD, MD, Francis X	Southside Va	BURGER, MD, Wilbur France	Lynchburg		
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BONES, MD, Jos Thos	Richmond	BRITT JR., MD, John Mills	Tri-County	BURNETT, MD, Gerald Crain	Halifax		
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BOOKER III, MD, James Judson	Southwest Va	BROADADDUS JR., MD, Carl A	Northern Neck	BURSLEM JR., MD, Wm Ashworth	Northern Va		
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BOOKER, MD, J Motley	Northern Neck	BROCK, MD, Jay David	Fredericksburg	BURTON, MD, Ashby Jeffries	Residents		
BOON, MD, Franklin F	Richmond	BROCK, MD, Lee Richard	Fredericksburg	BURTON, MD, Calvin Thos	Roanoke		
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BOOTH, MD, Jerry Clark	Richmond	BROOCKER, MD, Warren Alan	Richmond				
BOOTH, MD, Orin Watts	Newport News						

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CARMICHAEL, MD, Paul A	Roanoke	CHEN, MD, Cheng Nan	Alexandria	COCKER JR., MD, William Luther	Hampton	CRAWFORD JR, MD, Wm	Virginia Beach
CARMINES, MD, Fay Ashton	Newport News	CHEN, MD, Chun-Ming	Portsmouth	COLE JR., MD, John	Roanoke	CRAWFORD, MD, David J	Augusta
CARNEY, MD, David Anthony	Newport News	CHEN, MD, Jen-Wen	Richmond	COLE, MD, Dean Baldwin	Richmond	CRAWFORD, MD, John Custis	Out Of State
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CARPENTER, MD, Earnest B	Richmond	CHENG, MD, Chin Lin	Wise	COLE, MD, Ralph J	Arlington	CREEP, MD, J W	Chesapeake
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CARR JR, MD, Fay I	Portsmouth	CHESLER, MD, David L	Albemarle	COLEMAN, MD, Ashby	Lynchburg	CRETEUR, MD, Christian E	Portsmouth
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CARROLL, MD, Geo Jos	Fairfax	CHILDRESS JR, MD, A Jack	Norfolk	COLEMAN, MD, Wm P	Out Of State	CRIMM, MD, Carl Eugene	Fairfax
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CARROLL, MD, Robert P	Out Of State	CHILDREY JR, MD, Edgar	Richmond	COLL, MD, Jose D	Richmond	CRITTENDEN, MD, David Gray	Richmond
CARRON JR, MD, Harold	Albemarle	CHILDREY, MD, Stephan	Southside Va	COLLETTI, MD, Nicholas George	Prince William	CROCK, MD, Thomas Rankin	Fairfax
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CARTAGENA, MD, Rodolfo S	Wise	CHILES III, MD, Morton Perrin	Culpeper	COLLIER, MD, John E	Richmond	CROCKFORD, MD, Jon Lee	Norfolk
CARTER II, MD, B Noland	Richmond	CHISHOLM, MD, Louis Randolph	Richmond	COLLINS JR, MD, Phil	Albemarle	CROOKS JR, MD, Lewis Dani	Richmond
CARTER JR, MD, Hill	Richmond	CHITNUM, MD, Susan L	Residents	COLLINS, MD, Charles David	Tazewell	CROSBY, MD, Ivan Keith	Albemarle
CARTER JR, MD, Wilbur B	Halifax	CHITWOOD, MD, James Logan	Southwest Va	COLLINS, MD, Edward Frank	Out Of State	CROSBY, MD, James Foster	Chesapeake
CARTER JR, MD, William Herman	Fairfax	CHITWOOD, MD, Sarah E Roberts	Southwest Va	COLLIS, MD, Peter B	Alexandria	CROSETT JR, MD, Alexander D	Northern Neck
CARTER, MD, Bruce Thomas	Albemarle	CHITWOOD, MD, Walter R	Southwest Va	COLLMANN, MD, Warren X	Southside Va	CROSIER, MD, Joseph L	Southside Va
CARTER, MD, Edward Kent	Out Of State	CHIU, MD, Ming Sung	Southside Va	COMBS JR, MD, Luke	Albemarle	CROSS, MD, James Allerton	Wise
CARTER, MD, Henry G	Norfolk	CHMIEL, MD, Andrew Jos	Fairfax	COMBS, MD, Allen Evans	Newport News	CROSS, MD, James Parker	Norfolk
CARTER, MD, Robert Lee	Fairfax	CHOHANY, MD, Geo J	Williamsburg	COMER JR, MD, James Edward	Roanoke	CROSS, MD, John Armstrong	Newport News
CARTER, MD, Russell H	Norfolk	CHOI, MD, Chung Shin	Fairfax	COMER, MD, Francis E	Rockingham	CROSS, MD, John Earle	Northern Va
CARTER, MD, Wesley Byrd	Richmond	CHOI, MD, Koo Young	Fairfax	COMUNALE, MD, R A	Fairfax	CROSS, MD, Leland L	Albemarle
CARTWRIGHT, MD, Crosby W	Southside Va	CHOI, MD, Walter Sik	Hampton	CONANT, MD, Roger	Southwest Va	CROSS, MD, Phyllis De Carlo	Alexandria
CARTY JR, MD, James Walker	Norfolk	CHOU, MD, Yi-Nan	Alexandria	CONCA, MD, Dominick Michael	Augusta	CROSS, MD, Steven W	Residents
CARUSO, MD, Peter Virginus	Fairfax	CHOUHURRY, MD, Ali Azam	Southside Va	CONCORDERA, MD, Joseph A	Southside Va	CROSSEN, MD, Richard Wm	Richmond
CARWELL JR, MD, Glenn Ray	Virginia Beach	CHOUGH, MD, Daa Been	Portsmouth	CONELLY, MD, Lawrence J	Fredricksburg	CROSSLAND JR, MD, Clem C	Tri-County
CASABONA, MD, Albert C	Fairfax	CHOY, MD, Yoon Keun	Portsmouth	CONLEY, MD, Eugene Jos	Alexandria	CROSSLAND, MD, Stanley G	Arlington
CASEY, MD, Catherine Sue	Arlington	CHRISTIAN, MD, Chas Fletcher	Richmond	CONNER, MD, Alvin Eugene	Prince William	CROTEAU, MD, Louis John	Virginia Beach
CASEY, MD, Wm C	Fairfax	CHRISTIAN, MD, George Henry	Norfolk	CONQUEST, MD, H Fairfax	Richmond	CROUCH JR, MD, Earl Russell	Norfolk
CASHION, MD, Donald T	Norfolk	CHRISTIAN, MD, Wm E	Southwest Va	CONRAD, MD, Frederick Ellison	Southwest Va	CROW JR, MD, William Cecil	Roanoke
CASPARI, MD, Richard B	Richmond	CHRISTIE, MD, Laurence Glenn	Richmond	CONSTABLE, MD, Wm Chas	Albemarle	CROWDER JR, MD, Chas H	Southside Va
CASSADA, MD, Wm Abraham	Roanoke	CHU, MD, Young-Kwon	Virginia Beach	CONSTANT JR, MD, Tony	Richmond	CROWDER JR, MD, Thos Harold	Halifax
CASSIDY, MD, R L	Norfolk	CHUCKER, MD, Geo N	Allegheny	CONTIS, MD, George Peter	Arlington	CROWDER JR, MD, Robert V	Lynchburg
CASSIDY, MD, William Michael	Fairfax	CHUN, MD, Yong-Kwon	Tazewell	CONWAY, MD, Brian Peter	Albemarle	CROWDER, MD, Margaret E	Southside Va
CASTANEDA, MD, Albarto J	Chesapeake	CHUNG III, MD, Jey-Dea	Southwest Va	CONWAY, MD, Kevin	Roanoke	CROWDER, MD, Patricia Elliott	Orange
CASPER, MD, Louis J	Roanoke	CHUNG, MD, Doo Hyoun	Fairfax	COOGAN, MD, E A	Fairfax	CROWE, MD, Walter Geo	Fairfax
CASTILLEJO, MD, Raymundo A	Chesapeake	CHUNG, MD, Kyung Yil	Alexandria	COOK III, MD, John H	Northern Va	CROWLEY, MD, Junius Elliott	Roanoke
CASTILLO JR, MD, Vincente A	Stuart	CHURCH, MD, David Russell	Out Of State	COOK JR, MD, Wm A	Lynchburg	CRUIKSHANK, MD, Dwight P	Richmond
CASTILLO, MD, Probo H	Tazewell	CHUSID, MD, Aram	Fairfax	COOK JR, MD, William Henry	Southwest Va	CRUM, MD, Jerry Brice	Roanoke
CASTLE, MD, James Richard	Roanoke	CHUSUEI, MD, Richard V	Fairfax	COOK, MD, Chas Barrie	Fairfax	CRUSER, MD, Fred S	Roanoke
CASTLE, MD, Robert Lewis	Fairfax	CICCONI, MD, Alvin Jacob	Norfolk	COOK, MD, Gary Ruane	Fairfax	CSATARY, MD, Laszlo K	Arlington
CASTRO, MD, Ernesto V	Fairfax	CIGTAY, MD, Attila Sakir	Alexandria	COOK, MD, Irving Kenneth	Augusta	CUBE, MD, Ernesto Milla	Southwest Va
CATALANO, MD, Charles J	Lynchburg	CILLEY, MD, Richard D	Williamsburg	COOK, MD, J. Bryon	Culpeper	CUDWORTH, MD, Geo Hitchon	Roanoke
CATALDO, MD, Jos Richard	Alexandria	CIMMINO, MD, Christian V	Fredricksburg	COOKE, MD, Charles Lee	Richmond	CUESTA, MD, Maximo Lopez	Roanoke
CATE, MD, L Huntley	Culpeper	CIOFALO, MD, Carol Ellen	Arlington	COOKE, MD, Saml L	Richmond	CUI, MD, Marie Poldi	Hampton
CATES, MD, Michael A	Albemarle	CIOLETTI, MD, Roy Robert	Prince William	COOKSEY, MD, Wm Perry	Norfolk	CULLANDER, MD, Cecil C.H.	Albemarle
CATES, MD, Robert Judson	Fairfax	IRIC, MD, Andrew M	Virginia Beach	COOLEY, MD, Carl Conrad	Richmond	CULLER JR, MD, Richard L	Norfolk
CATLETT, MD, John B	Richmond	CLAGUE, MD, Allen Manville	Roanoke	COONE, MD, Herbert W	Prince William	CUMBA, MD, Jesse W	Albemarle
CATRON JR, MD, Stuart H	Southwest Va	CLAIBORNE JR, MD, Herbert A	Richmond	COONEY, MD, Dorothy F	Southside Va	CUMMINGS, MD, Charles Edward	Richmond
CAUGHRON, MD, Samuel Dan	Albemarle	CLAPP JR, MD, Henry W	Norfolk	COOPER JR, MD, Geo	Albemarle	CUMMINGS, MD, Wm Stevan	Williamsburg
CAULKINS JR, MD, Chas W	Augusta	CLAPP, MD, Deborah G	Fairfax	COOPER, MD, Alan Michael	Lynchburg	CUNDIFF, MD, David M	Norfolk
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CAULKINS, MD, Pamela M	Residents	CLARE, MD, Frank B	Portsmouth	COOPER, MD, John A D	Fairfax	CUNNINGHAM, MD, James K	Northern Neck
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CAVEDO JR, MD, Irvin Walters	Out Of State	CLARK, MD, Joe Lynn	Lynchburg	COPPEDGE, MD, Chas Wm	Prince William	CURTIS JR, MD, Walter	Hampton
CAVENDER, MD, Wm Francis	Alexandria	CLARK, MD, Laurence J	Alaxandria	COPPOLA, MD, Armando Ralph	Southside Va	CURWEN, MD, Geoffrey Wm	Patrick-Henry
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CECCHINO, MD, Robert Andrew	Virginia Beach	CLARKE JR, MD, Thos Hal	Southwest Va	COREN, MD, Sidney W	Virginia Beach		
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CHALASANI, MD, Srirama Prasad	Norfolk	CLARY, MD, Richard M	Richmond	COSTA, MD, Jack M	Fairfax		
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CHAMBERLAIN, MD, Richard R	Roanoke	CLEMENTS JR, MD, Ernest L	Richmond	COUK, MD, David Edgar	Fauquier		
CHAMBERS, MD, Beverly Noe	Northern Va	CLEMENTS, MD, Boyd M	Mid-Tidewater	COUK, MD, Macon Smiley	Richmond		
CHAMBERS, MD, Donald Edwin	Norfolk	CLEMENTS, MD, Francis J	Mid-Tidewater	COUPER, MD, John Lee	Rockbridge		
CHAN, MD, James C M	Richmond	CLIFT, MD, John Vinton	Fauquier	COURTNEY JR, MD, C.B.	Newport News		
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CHANDLER, MD, James Gilbert	Albemarle	CLINGENPEEL, MD, J Floyd	Tri-County	COVER, MD, Jesse R	Fairfax		
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CHAO, MD, Yu-Hua	Alexandria	CLOSE, MD, James Mc Clay	Fairfax	COWLING, MD, Lawrence Stanley	Newport News		
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CHAPMAN, MD, A Bredley	Alexandria	COBBLE, MD, Clerk Robert	Denville	COX, MD, James M	Lynchburg		
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CHARITY, MD, Cynthia M	Richmond	COFFELT, MD, Kenneth Clayton	Patrick-Henry	CRADDOCK, MD, Geo Barksdale	Lynchburg		
CHARITY, MD, Renard Adkins	Richmond	COFFEY, MD, E L	Roanoke	CRADDOCK, MD, Wm E	Albemarle		
CHARLTON, MD, James Perry	Virginia Beach	COHEN, MD, Alan Brent	Hampton	CRAGGS III, MD, Thomas F	Richmond		
CHARNEY, MD, David L	Alexandria	COHEN, MD, Alan Paul	Hampton	CRAIG III, MD, Seth Clayton	Fredricksburg		
CHASE, MD, Sandra Mae	Fairfax	COHEN, MD, Irwin Kelm	Southside Va	CRAIG, MD, James Wm	Albemarle		
CHAUDHARY, MD, Nazir Ahmad	Richmond	COHEN, MD, Joel Laurence	Danville	CRAMER, MD, Alfred Bartlett	Culpeper		
CHAUDHURI, MD, Moken Lal	Portsmouth	COHEN, MD, Lawrence	Loudoun	CRAMPTON, MD, Richard S	Albemarle		
CHAVEZ, MD, Rolando M.	Tazewell	COHEN, MD, Leonard Joel	Bedford	CRANTON, MD, Elmer Mitchell	Southwest Va		

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D'ALESSANDRO, MD, Frank T.	Albemarle
D'AMATO, MD, Nicholas Anthony	Norfolk
DABNEY JR, MD, Thos Todd	Richmond
DABNEY, MD, Wm Taylor	Richmond
DACHER, MD, Elliott S	Fairfax
DAFASHY, MD, Monir Y	Out Of State
DAGEFORD, MD, James R	Richmond
DAHM, MD, Norman Richard	Virginia Beach
DAIMLER, MD, John Charles	Newport News
DAJAO, MD, Rise Faith	Portsmouth
DAJAO, MD, Rogaciono M.	Chesapeake
DAKERMANDJI, MD, Farid	Allegheny
DALE, DO, James G	Northern Va
DALEY, MD, Timothy Horton	Arlington
DALEY, MD, Wm Edward	Fredricksburg
DALTON JR, MD, James B	Richmond
DALTON, MD, Henry Tucker	Arlington
DAMMANN, MD, John F	Albemarle
DAMRON, MD, Joseph McDonald	Northern Va
DANACEAU, MD, Henry Lawrence	Arlington
DANDRIDGE JR, MD, Wm Robt	Albemarle
DANDRIDGE, MD, Wm Robt	Albemarle
DANFORTH, DO, Michael Allen	Out Of State
DANFORTH III, MD, John M	Richmond
DANIEL JR, MD, Donald Sneed	Richmond
DANIEL, MD, Jergme M	Richmond
DANIEL, MD, John G	Residents
DANIEL, MD, Thos Moore	Richmond
DANIELS, MD, Warren C.	Southside Va
DARBY, MD, Daniel Lee	Portsmouth
DARDEN JR, MD, James Ryland	Richmond
DARDEN JR, MD, Oscar B	Bedford
DARNALL JR, MD, Robert A	Albemarle



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DART, MD, Robert C.	Fauquier	DEXTERS, MD, Yvonne L	Portsmouth	DUMLER JR., MD, John Chas	Rockingham	ELSASSER JR., MD, Geo F	Norfolk
DATOC, MD, Roberto L	Alexandria	DEYERLE, MD, Henry Price	Rockingham	DUMVILLE, MD, David Milton	Richmond	ELSBURG III, MD, Geo F	Alexandria
DAUBER, MD, N.C.	Culpeper	DEYERLE, MD, William Minor	Richmond	DUNCAN, MD, Victor Alberto	Out Of State	ELSWICK, MD, Ronald Kenneth	Southwest Va
DAUGHERTY, MD, Elizabeth C.	Fairfax	DEYTON, MD, Walter Edward	Danville	DUNDON, MD, Bruce Carroll	Virginia Beach	EMILIAN, MD, N.A.	Southside Va
DAUGHERTY, MD, Thomas W	Norfolk	DHILLON, MD, Avtar Singh	Residents	DUNDON, MD, Suzanne E	Norfolk	ENDE, MD, Milton Fapp	Southside Va
DAUGHTREY JR., MD, Walter F	Tri-County	DI FAZIO, MD, Cosmo Americo	Albemarle	DUNFORD, MD, Jos Leonard	Norfolk	ENG, MD, Benjamin Peter	Norfolk
DAUM, MD, Conrad Henry	Patrick-Henry	DI GIOVANNI JR., MD, Cleto	Residents	DUNMAN, MD, Lester Edwin	Southwest Va	ENGEL, MD, John Jos	Patrick Henry
DAVALOS, MD, Hugo A	Alexandria	DI PINTO, MD, Felix R	Alexandria	DUNN II, MD, Churchill Gibson	Loudoun	ENGH, MD, Charles A	Alexandria
DAVID, MD, Margalit Wynne	Richmond	DI SANDRO JR., MD, Giovanni	Fairfax	DUNN, MD, Harold Paul	Richmond	ENGH, MD, O Anderson	Arlington
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DAVID-NELSON, MD, Margit A	Alexandria	DIAMANT, MD, Hermann	Out Of State	DUNN, MD, Leo James	Richmond	ENSLIN, MD, Jassie Marsh	Lynchburg
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DAVIDSON, MD, Donald Dale	Richmond	DIAZ, MD, Dennis D	Mid-Water	DUNNINGTON, MD, Gansevoort H	Richmond	EPPESS III, MD, Edward M	Richmond
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DAVIE, MD, Steven Ames	Southwest Va	DICK, MD, Michael A	Rockbridge	DUNWIDDIE, MD, Walter C.	Residents	EPSTEIN, MD, Michael S	Residents
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DAVIES, MD, Linda High	Culpeper	DICKENS, MD, Michael Douglas	Fairfax	DURCAN, MD, Frank J	Northern Va	ERAGAN, MD, Mehmet Arif	Alexandria
DAVIS III, MD, John Edward	Northern Va	DICKENS, MD, Nandini	Fairfax	DURHAM, MD, Alfred A	Roanoke	ERASMO, MD, Ramon Rondobio	Danville
DAVIS IV, MD, John Staige	Albemarle	DICKERSON, MD, John Wm	Norfolk	DURICA, MD, David L	Portsmouth	ERBA, MD, S. Michael	Lynchburg
DAVIS JR., MD, Chas Emmett	Norfolk	DICKERSON, MD, Shelby Clark	Roanoke	DURKIN, MD, James Patrick	Arlington	ERDM, MD, Feyerz	Roanoke
DAVIS JR., MD, Chas Stanley	Virginia Beach	DICKINSON, MD, Clara R King	Roanoke	DURR JR., MD, Michael Jos	Fairfax	ERDMAN, MD, Robert L	Northampton
DAVIS JR., MD, Edward Garland	Richmond	DICKINSON, MD, Wm Andrew	Virginia Beach	DURRAN JR., MD, Waheed	Southside Va	ERICKSEN, MD, Thomas W.	Alexandria
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DAVIS JR., MD, Leonard Leslie	Portsmouth	DICKSON, MD, William Henry	Fairfax	DUTT, MD, Cyril Prabhakar	Out Of State	ERIM, MD, Zeki	Alexandria
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DAYANIM, MD, Behrooz	Portsmouth	DODD, MD, Richard Wine	Richmond			EVANS, MD, Cecil F	Newport News
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DE BUTTS, MD, Richard E	Portsmouth	DOE, MD, Frederick David	Out Of State			EVANS, MD, James Dewitt	Rockingham
DE LA CRUZ, MD, Gloria V	Norfolk	DOGAN, MD, M. Ezel	Fairfax			EVANS, MD, James Gregory	Virginia Beach
DE LAURIA, MD, Frank Anthony	Prince William	DOGRUL, MD, Suleyman S	Hampton			EVANS, MD, Martin Terry	Richmond
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DE VOCHT, MD, Ludovic Jules	Alexandria	DONLAN JR., MD, Charles J	Chesapeake			EVERS, MD, Jos Chas	Fairfax
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DEANS, MD, Robt Douglas	Southwest Va	DONOWITZ, MD, Gerald R	Albemarle			EWELL, MD, Murlin Knox	Albemarle
DEATON, MD, Richard Thos	Norfolk	DONOWITZ, MD, Leigh G	Albemarle			EWING, MD, Nathaniel C	Lae
DEBLASI, MD, Robt F	Fredericksburg	DOOLEY, MD, Mark C	Richmond				
DEBNATH, MD, Kiran Sankar	Portsmouth	DOOLEY, MD, Parker Clark	Accomack				
DEBS, MD, Anthony	Fairfax	DORON, MD, Isaac G	Tri-County				
DECKER, MD, Henry Chesley	Richmond	DORSEY III, MD, John Thomas	Residents				
DECKER, MD, Michael John	Richmond	DOS SANTOS III, MD, Jose G	Richmond				
DECKER, MD, Sterling Randolph	Virginia Beach	DOSHI, MD, Ramesh D	Southside Va				
DEE, MD, Paul Michael	Albemarle	DOSS, MD, Julian Booth	Roanoke				
DEEP, MD, Anthony Abraham	Richmond	DOSS, MD, Otis Wm	Northampton				
DEEP, MD, Wm Danl	Richmond	DOUGHERTY, MD, Clyde Hudson	Southside Va				
DEGEN, MD, Douglas B	Augusta	DOUGHERTY, MD, William E	Arlington				
DEIGNAN JR., MD, Jos Michael	Northern Va	DOWLING, MD, Jean Marie	Virginia Beach				
DEL PILAR, MD, Jaime V	Prince William	DOWNS, MD, Edward Jay	Chesapeake				
DEL PILAR, MD, Leticia C. Q.	Prince William	DOWNS, MD, Peter Ellsworth	Virginia Beach				
DEL SORDO, MD, Andrew A	Richmond	DOZORETZ, MD, Ronald Irving	Portsmouth				
DEL VECCHIO, MD, Michael A	Fairfax	DRAKE JR., MD, Charles R	Residents				
DELANEY JR., MD, Martin D	Alexandria	DRAKE, MD, John Edward	Richmond				
DELANEY, MD, Thomas J	Lynchburg	DRAFLER III, MD, Siegfried	Out Of State				
DELANEY, MD, Wm Morgan	Alexandria	DREIFUSS, MD, Fritz Emanuel	Albemarle				
DELAWEY, MD, Hilbert H	Albemarle	DREW, MD, Donald W	Norfolk				
DELLINGER, MD, James Lyle	Fauquier	DREW, MD, J. Edwin	Fairfax				
DELMORE, MD, Donald P	Alexandria	DREWRY, MD, David B	Southside Va				
DEMARI, MD, Lorenzo A	Richmond	DRIEBE, MD, W T	Arlington				
DEMIRAY, MD, Adel	Out Of State	DRISKILL JR., MD, Wm Lawson	Lynchburg				
DEMSEY, MD, William Charles	Fairfax	DRIVER, MD, Semi Francis	Roanoke				
DENARO JR., MD, Frank	Portsmouth	DRUCKER, MD, Jacob R	Norfolk				
DENGEL, MD, Gisela A	Northern Va	DRUMMOND JR., MD, Chas Stitt	Richmond				
DENIS, MD, Roger	Newport News	DRYSDALE, MD, Daniel B	Southwest Va				
DENIUS, MD, Larry Richard	Allegheny	DU BU, MD, Jean Bernard	Hampton				
DENT, MD, Roy Wm	Richmond	DU PREY, MD, Robt Edward	Northern Neck				
DERDEYN, MD, Andre Philip	Albemarle	DU PUY, MD, Theodore E	Chesapeake				
DERKAC, MD, Wayne M	Norfolk	DU ROCHER, MD, Frances A	Fairfax				
DERRING JR., MD, Eldridge H	Norfolk	DUCCI, MD, Hector H	Fairfax				
DESOUZA, MD, Romaldo F X	Alexandria	DUCEY, MD, Kevin Francis	Roanoke				
DESPER, MD, Paul Carlton	Prince William	DUCK, MD, George Bryan	Richmond				
DETHMERS, MD, Daniel A	Richmond	DUCKWALL, MD, Francis Jos	Roanoke				
DEUTSCH, MD, Alan Seth	Alexandria	DUCKWORTH, MD, Elizabeth H	Roanoke				
DEVEREUX, MD, James Peter	Norfolk	DUDLEY, MD, Frank Humbert	Roanoke				
DEVINE JR., MD, Chas Jos	Norfolk	DUFFY, MD, Adrian Dominick	Fairfax				
DEVINE, MD, Joan W	Virginia Beach	DUFFY, MD, Eileen Josephine	Fairfax				
DEVINE, MD, Patrick Campbell	Norfolk	DUGGAN, MD, Paul M	Fairfax				
DEVLIN, MD, James Timothy	Virginia Beach	DUGGINS, MD, Virginia A	Fairfax				



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FEINMAN, MD, Maxwell Carlton	Lynchburg	FOX, MD, Kurt Johannes	Rockbridge	GARDNER, MD, James L	Southwest Va	GLADSTONE, MD, Jos E	Northampton
FEKETE, MD, Andrew Maurice	Norfolk	FOX, MD, Nelson Moffett	Patrick-Henry	GARDNER, MD, John E	Roanoke	GLASGOW, MD, Jean M Martin	Roanoke
FELDMAN, MD, Daniel M	Oul Of State	FOX, MD, Parham R	Lynchburg	GARDNER, MD, Jos Erskin	Rockingham	GLAZIER, MD, Richard Lee	Richmond
FELDMAN, MD, Frances R	Norfolk	FRABLE, MD, Mary Ann Smith	Richmond	GARDNER, MD, Richard Eneisi	Augusta	GLEASON, MD, Chas Henry	Albemarle
FELTON III, MD, Warren L	Residents	FRAGLE, MD, Wm Jackson	Richmond	GARDNER, MD, Robt D	Lynchburg	GLEASON, MD, Margaret Daley	Richmond
FELTON, MD, Harold Wm	Mid-Tidewater	FRAGA, MD, Vivian Maria	Residents	GARDNER, MD, Shockey D	Richmond	GLENN, MD, Robt Lee	Lynchburg
FEMINELLA JR., MD, John Geo	Southside Va	FRAMM, MD, Danl Herschal	Fairfax	GARDNER, MD, Stephen R	Residents	GLICK JR., MD, John T	Rockingham
FENDER, MD, Gary R	Arlington	FRANCIS JR., MD, Cleveland	Alexandria	GARNER, MD, David S	Roanoke	GLICKMAN, MD, Marc Harris	Norfolk
FENDERSON, MD, Allen Rex	Virginia Beach	FRANCIS, MD, Jamison K	Prince William	GARNER, MD, Fredric Bruce	Fredericksburg	GLONTZ, MD, Gary Edwin	Roanoke
FENSTERER JR., MD, Philip H	Halifax	FRANCIS, MD, John A	Accomack	GARNER, MD, Wallace C	Newport News	GLOVER JR., MD, Wm Lloyd	Fairfax
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FERGUSON, MD, William	Portsmouth	FRANCO, MD, Paulo E	Fairfax	GARRETT, MD, James Ellis	Norfolk	GLUCK, MD, Gabriel	Prince William
FERLAZZO, MD, Stephen L	Prince William	FRANK, MD, Robert J	Newport News	GARRISON III, MD, Jos Shermar	Portsmouth	GLUCKMAN, MD, Jeffrey B	Newport News
FERNANDEZ, MD, Frank L	Richmond	FRANK, MD, William E	Lynchburg	GARRISON, MD, Bobby	Norfolk	GOALD, MD, Harold Jerome	Arlington
FERRAR, MD, Wm Lewis	Richmond	FRANKEL, MD, Charles J	Albemarle	GARRISON, MD, David D	Virginia Beach	GODWIN, MD, Gene Arthur	Roanoke
FERRELL JR., MD, Haskins	Alexandria	FRANKEL, MD, Nicholas	Prince William	GARRISON, MD, Jack Stiles	Virginia Beach	GODWIN, MD, Ira David	Fairfax
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FERRY JR., MD, Darwin John	Roanoke	FRANKLIN, MD, John	Norfolk	GARZON, MD, Fernando Luis	Southwest Va	GOLDBERG JR., MD, Marvin	Norfolk
FERRY, MD, Allen M	Arlington	FRANKLIN, MD, William George	Arlington	GASHI, MD, Faton	Fairfax	GOLDBERG, MD, Jay Stephen	Southside Va
FIDLER, MD, Robt Young	Richmond	FRANTZ, MD, John F	Newport News	GASPAR, MD, Maurice L	Arlington	GOLDBERG, MD, Michael Harvey	Fairfax
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FIELD, MD, Burton Eugene	James River	FRASER JR., MD, Douglas J	Fairfax	GATES, MD, Thomas Jarman	Loudoun	GOLDBLATT, MD, Seymour Z	Alexandria
FIELDS, MD, Richard Lee	Fairfax	FRATKIN, MD, Melvin Joal	Richmond	GATZEK, MD, Werner John	Augusta	GOLDENBERG, MD, Robin Ira	Fairfax
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FIERRO, MD, Anthony	Richmond	FRÄZER, MD, William Penn	Loudoun	GAUNT, MD, Hunter Marshall	Northern Va	GOLDIN, MD, Milton	Portsmouth
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FIERRO, MD, Robert J	Richmond	FRAZIER, MD, John Richard	Roanoke	GAYDOS, MD, Lawrence Allred	Arlington	GOLDMAN, MD, Chas Jay	Norfolk
FIFER, MD, Carson Lee	Alexandria	FRAZIER, MD, Maurice W	Hampton	GAYLE JR., MD, Wm Earle	Lynchburg	GOLDMAN, MD, Edwin E	Chesapeake
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FISCHER, MD, Geo L	Alleghany	FREUND, MD, Jack	Richmond	GEISERT, MD, Todd W	Northern Neck	GONDOR, MD, Leslie Paul	Alexandria
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FISHER, MD, Richard Harding	Roanoke	FRIEDLIS, MD, Mayo	Alexandria	GENTILE, MD, Francis G	Richmond	GONZALES, MD, Patricia D	Southside Va
FITCH, MD, Willard M	Richmond	FRIEDMAN, MD, Asher Arthur	Norfolk	GEOLY, MD, Kenneth Lucian	Fairfax	GONZALEZ, MD, Juan M	Southside Va
FITCHETT, MD, Claiborne W	Norfolk	FRIEDMAN, MD, Carl Jeffrey	Albemarle	GEORGE, MD, Edward Richard	Norfolk	GONZALEZ, MD, Miguel H	Alexandria
FITZ-HUGH, MD, G. Slaughter	Albemarle	FRIEDMAN, MD, Michael Harbert	Alexandria	GERDE, MD, Leland	Williamsburg	GONZALEZ, MD, Ullyses S	Wise
FITZER, MD, Peter Malcolm	Newport News	FRIERSON JR., MD, John Hugh	Richmond	GERGEN, MD, Werner Anthony	Out Of State	GOOCH III, MD, Garrett G	Roanoke
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FITZGERALD, MD, Paul Francis	Lynchburg	FRIITZ, MD, Robert L	Albemarle	GHAEMI III, MD, Kamal	Fairfax	GOODE JR., MD, Harvey W	Northern Neck
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FOGLE, MD, Kelly Ashworth	Richmond			GILL, MD, Fleming W	Richmond	GOTTICO, MD, Rustico Tolentino	Augusta
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GREENBERG, MD, Milton	Danville			HARRIS JR, MD, James Campbell	Richmond	HENRY JR, MD, Lester F	Hampton
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GREER, MD, Wm Crockett	Roanoke			HARRIS, MD, Robt Eldred	Out Of State	HERMANSEN, MD, Karen L.	Fairfax
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GREEVER, MD, Wm N	Southwest Va			HARRIS, MD, Ronald B	Roanoke	HERNANDEZ, MD, Manuel O	Fairfax
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GREGORIOU, MD, Panos Geo	Buchanan			HARRISON III, MD, Carrington	Augusta	HERR JR, MD, Austin Alexis	Alexandria
GREGORY, MD, James Stewart	Portsmouth			HARRISON, MD, H. Courtenay	Virginia Beach	HERRING JR, MD, Alvah L	Southside Va
GREGORY, MD, Roger Thorpe	Richmond			HARRISON, MD, Henry Tucker	Richmond	HERRING JR, MD, Russell E	Patrick-Henry
GREGORY, MD, Warren C	Norfolk			HARRISON, MD, Jacquelin M	Richmond	HERRING, MD, Angela	Newport News
GRELLA JR, MD, Benjamin	Northern Va			HARRY, MD, Frederick P.	Southside Va	HERRINGTON, MD, Marvin S	Norfolk
GREMER, MD, John Saml	Southside Va			HARRY, MD, Robert Roger	Fredericksburg	HERMAN, MD, Joanne	Fairfax
GRESINGER, MD, Thomas Hamlin	Newport News			HARSANYI, MD, Paul Gabor	Fredericksburg	HERRON, DO, Robert Thomas	Fairfax
GRETES, MD, John C	Fairfax			HARSHAW JR, MD, William Geo	Fairfax	HERSH, MD, Stephen Robt	Alexandria
GRETHNER, MD, Eugene Rudolf	Alexandria			HART JR, MD, Kirby Thompson	Southside Va	HERSHBERG, MD, Sandra Gail	Fairfax
GREY, MD, Wm Hugh	Augusta			HART JR, MD, Richard Jos	Fairfax	HERSHEY, MD, J. Henry	Residents
GRIER III, MD, Geo S	Newport News			HARTER, MD, Basil T	Out Of State	HERTZBERG, MD, Michael	Alexandria
GRIFFEY, MD, Richard Thos	Norfolk			HARTLEY, MD, A. Howland	Richmond	HESS, MD, Chas Edwin	Albamarle
GRIFFIN JR, MD, Harvey Lee	Southside Va			HARTMAN, MD, Carl Wm	Norfolk	HESS, MD, Irvin Eugene	Rockingham
GRIFFIN JR, MD, Edward E.	Out Of State			HARTWRIGHT, MD, Alva James	Hampton	HESS, MD, James Brock	Williamsburg
GRIFFIN, MD, Francis G	Virginia Beach			HARVAN, MD, David	Fairfax	HESS, MD, John Milton	Norfolk
GRIFFIN, MD, Gary K.	Residents			HARVEY, MD, Chas Thos	Danville	HESS, MD, Leonard Wayne	Out Of State
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GRIGSBY JR, MD, Wm C	Out Of State			HASHEM, MD, Abul	Wide	HEYNER, MD, Gregory James	Richmond
GRIGSBY, MD, Wm Paul	Scott			HASKELL JR, MD, Edward G	Norfolk	HICKAM, MD, George Lindsay	Roanoke
GRIM, MD, James Franklin	Fairfax			HASKO, MD, Barbara Ann	Fairfax	HICKMAN, MD, Clifton C.	Richmond
GRINNAN JR, MD, R Bryan	Norfolk			HASSAN, MD, Meborah	Arlington	HICKMAN, MD, Janet Grater	Lynchburg
GRINNAN, MD, Geo Lamb Buist	Norfolk			HASSLER, MD, Carol R.	Albamarle	HICKMAN, MD, Robert E	Lynchburg
GRINNAN, MD, Richardson	Richmond			HASTILLO, MD, Andrea Karen	Richmond	HICKSON, MD, Edward Watts	Lynchburg
GRINNAN, MD, Wm C	Richmond			HATCHER, MD, Wm F	Roanoke	HIGGINS JR, MD, Wm Harrison	Richmond
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GRISWOLD, MD, James Francis	Virginia Beach			HATFIELD, MD, Wm Henry	Southwest Va	HIGGINS, MD, Michael R.	Norfolk
GRISWOLD, MD, Martha A.	Southside Va			HATTEN, MD, John Q	Newport News	HIGGS, MD, James Albert	Augusta
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GROSS JR, MD, Frederick M	Fairfax			HAUSER, MD, Walter Arthur	Southside Va	HILL, MD, David Bennett	Lynchburg
GROSS, MD, Alton F	Danville			HAUT, MD, Donald David	Alexandria	HILL, MD, Dougless Orville	Northern Va
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GROUSE, MD, Lawrence Douglas	Out Of State			HAWKINS, MD, Richard F	Southwest Va	HILL, MD, Kathryn Dodds	Virginia Beach
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GUIRGUIS, MD, Habib Habib	Richmond			HEIDE, MD, Robert Kay	Albamarle	HODGES, MD, Thos O	Out Of State
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HOKE, MD, Harry Franklin	Richmond	HULVEY, MD, J Thomas	Southwest Va	JANES JR, MD, John R.	Richmond	JORDAN, MD, Wm R	Richmond
HOLCOMB III, MD, Harry Sherman	Northampton	HUMPHREY, MD, William Trowell	Norfolk	JANSEN, MD, George Allen	Out Of State	JOSE, MD, Nora D	Alexandria
HOLDEN, MD, Bobby G	Southside Va	HUMPHRIES III, MD, Marion K	Roanoke	JARBADAN, MD, Ignaz Papa	Prince William	JOSE, MD, Pedro A	Alexandria
HOLLAND, MD, Clarence Adrian	Virginia Beach	HUMPHRIES JR., MD, Marion K	Albemarle	JARRELL, MD, Shelby Edward	Richmond	JOSEPH-GUANZON, MD, Pat	Danville
HOLLAND, MD, Henry Davis	Richmond	HUMPHRIES JR., MD, William H	Roanoke	JARRETT, MD, Alvin Quarles	Virginia Beach	JOSEPHIAK, MD, Eugene Jos	Roanoke
HOLLAND, MD, Walter R	Lynchburg	HUMPHRIES, MD, Thos J	Roanoke	JARRETT, MD, Harry Walthall	Lynchburg	JOSEPH, MD, Charles R	Lynchburg
HOLLAND, MD, William Elisha	Richmond	HUMPHRIES, MD, Wm C	Richmond	JARRETT, MD, John Tallman	Richmond	JOSEPHTHAL, MD, Danl Herbert	Albemarle
HOLLINGSWORTH, MD, John H	Roanoke	HUNDLEY, MD, Jos Leigh	Out Of State	JASON, MD, Casey John	Fairfax	JOSHUA, MD, Alan	Fairfax
HOLLIS, MD, Joseph B	Portsmouth	HUNNICUTT, MD, Thomas W.	Out Of State	JASTRZESKI, MD, George W	Arlington	JOUBIN, MD, Jahan M	Prince William
HOLLISTER JR., MD, Wm	Fredericksburg	HUNT, MD, Chas F	Richmond	JAVATE, MD, Rosy T	Arlington	JOYCE, MD, Judith Marie	Residents
HOLLOWELL, MD, John W	Richmond	HUNT, MD, Patricia A	Richmond	JEAN, MD, Cheo Ming	Southwest Va	JOYNER, MD, Raymond Kenneth	Hampton
HOLMES, MD, Francis Hammond	Portsmouth	HUNT, MD, Robt Clarence	Fairfax	JEAN-GILLES, MD, Brunet	Norfolk	JOYNES, MD, Michael Hope	Hampton
HOLMES, MD, I Earl	Augusta	HUNTER, MD, James Gordon	Lynchburg	JEDEIKIN, MD, Roy	Albemarle	JUDSON, MD, Saml Benji	Arlington
HOLSINGER, MD, Donald Rider	Patrick-Henry	HUNTER, MD, Richard Grant	Albemarle	JEFFERIES, MD, Allan H	Norfolk	JUDY, MD, Saml Benji	Southside Va
HOLSINGER, MD, James R	Northern Va	HUNTER, MD, Samuel B	Richmond	JEFFREY, MD, M. Graves	Norfolk	JURS, MD, Dennis Gregg	Southwest Va
HOLT JR., MD, Mark Edgar	Hampton	HUNTER, MD, Thos Harrison	Albemarle	JENKINS, MD, Chas E	Alexandria	JUSAY, MD, Felciano J	Southwest Va
HOLT JR., MD, Frank Loving	Residents	HUNTER, MD, William Mills	Hampton	JENKINS, MD, Daniel	Williamsburg		
HOLT, MD, Bevel Dan	Out Of State	HUNTINGTON, MD, Daniele F	Fairfax	JENNETTE, MD, Arthur Harris	Norfolk		
HOLT, MD, Ronald R	Fairfax	HURT JR., MD, John Omohundro	Roanoke	JENNINGS JR., MD, C Leon	Roanoke		
HOLTHAUS, MD, Wallace Harold	Rockingham	HURT, MD, Alvin Judson	Roanoke	JENNINGS JR., MD, Rutus B	Norfolk		
HOLYFIELD, MD, Paul Altred	Patrick-Henry	HURT, MD, Geo Adams	Roanoke	JENNINGS, MD, Eileen Thorpe	Bedford		
HOMA, MD, Michael	Arlington	HURT, MD, Waverly Glenn	Lynchburg	JENNINGS, MD, Robt Hutchings	Albemarle		
HONEYCUTT JR., MD, Grover C	Scott	HURTADO, MD, Rodrigo C	Richmond	JENNINGS, MD, Thornton S	Out Of State		
HONG, MD, Seung Kook	Arlington	HURWITZ, MD, Byron Stuart	Fairfax	JENNINGS, MD, Thos H	Bedford		
HONG, MD, Young Sung	Hampton	HURWITZ, MD, Richard L	Norfolk	JENNINGS, MD, W. Stanley	Chesapeake		
HOOK JR., MD, Edward W	Albemarle	HUTCHER, MD, Neil Edward	Richmond	JENNETTE, MD, Freeman Wesley	Bedford		
HOOPER, MD, Herbert C	Southside Va	HUTCHESON JR., MD, Robt S	Roanoke	JENSEN, MD, Edward Walter	Southside Va		
HOOPER, MD, Roy Michael	Out Of State	HUTCHESON JR., MD, Jack R	Roanoke	JEROY, MD, Harry Keirn	Norfolk		
HOOPER, MD, Wm	Norfolk	HUTCHESON, MD, Janet	Arlington	JESNECK, MD, Edward	Patrick-Henry		
HOPEWELL, MD, Edward Lee	Northern Va	HUTCHISON, MD, Wayne Thomas	Bedford	JESSEE JR, MD, Edgar Forrest	Richmond		
HOPKINS JR., MD, John David	Norfolk	HUTTON, MD, Chas Frederick	Richmond	JESSEE, MD, Robt W	Richmond		
HOPKINS, MD, Jay Everett	Lynchburg	HYLTON JR., MD, Paul Hampton	Northern Va	JESSEE, MD, Samuel D.	Richmond		
HOPWOOD, MD, Herbert G	Arlington	HYLTON, MD, Claude Kavilla	Prince William	JESSUP, MD, Douglas E	Richmond		
HOQ, MD, Kasedul	Portsmouth	HYLTON, MD, James Moir	Southwest Va	JETER, MD, William Richard	Richmond		
HORAN, MD, Michael Thomas	Fairfax	HYSLIP, MD, John Wesley	Richmond	JEWUSIAK, MD, Edward M	Williamsburg		
HORDEN, MD, Harold Milton	Norfolk			JOHN, MD, Sarah A	Fairfax		
HORGAN, MD, John A	Newport News			JOHNS, MD, Michael Edward	Out Of State		
HORN, MD, Henry J	Fairfax			JOHNS, MD, Thos Nelson Page	Richmond		
HORN, MD, Martin Seth	Fairfax			JOHNS, MD, Thos R	Albemarle		
HORNE, MD, Allen Bernard	Fairfax			JOHNS, MD, Wm A	Richmond		
HORNE, MD, Francis G	Out Of State			JOHNSON III, MD, Charles M	Albemarle		
HORNE, MD, Melvin L	Out Of State			JOHNSON JR., MD, Lester Dean	Fairfax		
HORNEY, MD, Wayne D	Southwest Va			JOHNSON JR., MD, Marriot C	Fredericksburg		
HORNG, MD, Fang Shuh	Northern Va			JOHNSON JR., MD, Walter W	Alleghany		
HORNBSBY, MD, Robert P	Southwest Va			JOHNSON, MD, Alfred G	Southside Va		
HOROWITZ, MD, Jed H	Residents			JOHNSON, MD, Allen C	Southside Va		
HORSCH, MD, Robert F	Southwest Va			JOHNSON, MD, Bruce E	Richmond		
HORSLEY, MD, John S	Richmond			JOHNSON, MD, Bruce E	Virginia Beach		
HORTENSTINE, MD, John C	Northern Va			JOHNSON, MD, Burton Allan	Fairfax		
HORTON, MD, Chas Edwin	Norfolk			JOHNSON, MD, David H	Norfolk		
HORTON, MD, Jack Donald	Fairfax			JOHNSON, MD, David Lawis	Fredericksburg		
HOSFIELD, MD, Richard H	Mid-Tidewater			JOHNSON, MD, Earl Robt	Roanoke		
HOSFIELD, MD, Wm Howard	Mid-Tidewater			JOHNSON, MD, Eileen El Dorado	Lynchburg		
HOSHINO, MD, David Ken	Accomack			JOHNSON, MD, Frank Mitchell	Roanoke		
HOSKINS, MD, Horace D	Danville			JOHNSON, MD, Geo Wm	Richmond		
HOSSAIN, MD, Mohammad A	Norfolk			JOHNSON, MD, Hal S	Richmond		
HOSTLER, MD, Sharon Lee	Albemarle			JOHNSON, MD, Harry I	Roanoke		
HOTCHKISS, MD, Wm J	Rockingham			JOHNSON, MD, John Walter	Northern Neck		
HOTCHKISS, MD, Wm S	Norfolk			JOHNSON, MD, Kenneth Roger	Alexandria		
HOUCK JR., MD, Wm Albert	Northern Va			JOHNSON, MD, L. Meredith	Out Of State		
HOUCK, MD, Jos W	Lynchburg			JOHNSON, MD, Wada Lane	Newport News		
HOUSER, MD, Peter Wm	Richmond			JOHNSON, MD, Walter Smith	Roanoke		
HOVLAND, MD, William Neal	Norfolk			JOHNSON, MD, Warren E	Loudoun		
HOWARD JR., MD, Robt Edwin	Hampton			JOHNSON, MD, William Waldo	Out Of State		
HOWARD, MD, Hall Rentro	Williamsburg			JOHNSON, MD, Wm Rayner	Northern Va		
HOWARD, MD, Lawrence Max	Lynchburg			JOHNSON, MD, Wm Thos	Virginia Beach		
HOWARD, MD, Robt L	Fairfax			JOHNSON, MD, Chas L	Richmond		
HOWE JR., MD, Allen K	Fairfax			JOHNSTON JR., MD, Elizabeth W	Fairfax		
HOWE, MD, James Robt	Alexandria			JOHNSTON, MD, Elizabeth W	Patrick-Henry		
HOWELL JR., MD, Edgar V	Out Of State			JOHNSTON, MD, John Dorrens	Tazewell		
HOWELL, MD, Donald S	Tri-County			JOHNSTON, MD, Mary E	Northern Va		
HOWELL, MD, Hannibal Eldredge	Hampton			JOHNSTON, MD, Randolph Page	Norfolk		
HOWELL, MD, Hugh Richard	Richmond			JOHNSTON, MD, Russell G	Richmond		
HOWELL, MD, Talmadge Rudolph	Southside Va			JOHNSTON, MD, Wm Burton	Richmond		
HOWERTON, MD, James Robert	Newport News			JOHNSTONE, MD, William T	Lynchburg		
HOWLETT, MD, Stephen Andrew	Augusta			JONAS, MD, John F	Alexandria		
HOWREN JR., MD, Harry H	Richmond			JONES JR, MD, Benj C	Richmond		
HOWZE, MD, Herbert H	Wise			JONES JR, MD, Beverly	Norfolk		
HOYLE, MD, John D	Alexandria			JONES JR, MD, Brock Darden	Albemarle		
HOYME, MD, Jane C	Arlington			JONES JR, MD, Herbert C	Richmond		
HOYT, MD, Robert Eugene	Northern Neck			JONES JR, MD, Warren Jeffrey	Norfolk		
HRANOWSKY, MD, Nicholas P	Richmond			JONES JR, MD, Wm Russell	Richmond		
HU, MD, Anthony Wen-Shih	Southside Va			JONES JR, MD, Howard Wilbur	Norfolk		
HUANG, MD, Amy Hwei-Mei	Fairfax			JONES, MD, Basil B	Richmond		
HUBACH, MD, Frederick Willis	Fairfax			JONES, MD, Benj Newman	Roanoke		
HUBBARD II, MD, George W.	Norfolk			JONES, MD, Daniel Ralph	Roanoke		
HUBER, MD, Albert Leopold	Albemarle			JONES, MD, Delmas Bernard	Wise		
HUBER, MD, Chas Mac	Northern Va			JONES, MD, Donald Kenneth	Richmond		
HUBERMAN, MD, Richard	Alexandria			JONES, MD, Edward A	Fairfax		
HUBERT, MD, Juergen	Richmond			JONES, MD, Geo Robt	Richmond		
HUCKE, MD, Andrew M.	Albemarle			JONES, MD, George Frederick	Southwest Va		
HUDGENS, MD, Robt Oscar	Richmond			JONES, MD, Georgeanna Seegar	Norfolk		
HUDGINS, MD, Earl Maxwell	Richmond			JONES, MD, Gordon Willis	Fredericksburg		
HUDGINS, MD, Hubert Bland	Mid-Tidewater			JONES, MD, J. Kipling	Southside Va		
HUDSON, MD, Chas A	Alexandria			JONES, MD, James Barrett	Lynchburg		
HUDSON, MD, Gwendolyn S	Richmond			JONES, MD, John P	Richmond		
HUDSON, MD, Sue Beth	Virginia Beach			JONES, MD, John Paul	Newport News		
				JONES, MD, John R	Richmond		
				JONES, MD, Joseph L.	Williamsburg		



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KELLS, MD, Douglas U	Tri-County	KISHORE, MD, P R S	Richmond			LEE, MD, Hyung Mo	Richmond
KELLY III, MD, John Jackson	Richmond	KISTLER, MD, Philip Crosby	Roanoke			LEE, MD, John Edward	Southwest Va
KELLY JR, MD, Frank R	Richmond	KISTNER, MD, James Robert	Albemarle			LEE, MD, Kok Seah	Prince William
KELLY JR, MD, Geo W	Southwest Va	KITCES, MD, Eileen C	Richmond			LEE, MD, Kyung Ja Shin	Fairfax
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KELLY JR, MD, Timothy L	Arlington	KITTERMAN, MD, James S	Norfolk			LEE, MD, Margaret C	Alexandria
KELLY, MD, John Francis	Fairfax	KLAM, MD, Warren Peter	Fairfax			LEE, MD, Maria E	Norfolk
KELLY, MD, Thaddeus Elliott	Albemarle	KLAPPROTH, MD, Hans Joachim	Fairfax			LEE, MD, Ming S	Southside Va
KELSEY, MD, Gerdi D	Virginia Beach	KLEDZIK, MD, Ronald Bruce	Virginia Beach			LEE, MD, Ralph Navero	Newport News
KELSEY, MD, Ronald Leon	Fredericksburg	KLEIN, MD, Arthur	Richmond			LEE, MD, Richard Mimms	Hanover
KEMP JR, MD, Verbon Eric	Richmond	KLEIN, MD, Frederick A	Richmond			LEE, MD, Roberto Juan	Southwest Va
KENDALL, MD, Robert Gentry	Northern Va	KLEMMER, MD, Philip John	Northern Va			LEE, MD, Sang Nam	Fairfax
KENDERS, MD, Kathryn L	Fairfax	KLIM, DO, Philip A	Rockingham			LEE, MD, Soo Ik	Albemarle
KENDIG JR, MD, Edwin L	Richmond	KLIMOCK, MD, Gregory	Northern Neck			LEE, MD, Sun Geun	Richmond
KENDRICK JR, MD, John Fox	Richmond	KLOTZ JR, MD, Jeremiah A	Norfolk			LEE, MD, Thaddeus Carmichael	Southwest Va
KENDRICK, MD, Marvin H	Alexandria	KLOUSIA, MD, John Walter	Alexandria			LEE, MD, Won Ro	Fairfax
KENERSON, MD, John G	Virginia Beach	KLUGE, MD, Robt Carter	Augusta			LEE, MD, Young	Southside Va
KENLEY, MD, James B	Richmond	KNAPP, MD, Robt Woodruff	Portsmouth			LEET, MD, Christopher J	Prince William
KENNAN JR, MD, Richard B	Out Of State	KNARR, MD, John Weidner	Southwest Va			LEFFKE, MD, David Wm	Lynchburg
KENNAN, MD, Thos F	Rockbridge	KNauft, MD, Richard David	Portsmouth			LEFRANK, MD, Edward Arthur	Fairfax
KENNEDY, MD, Carol Elizabeth	Fairfax	KNAYSII, MD, George A	Richmond			LEGASPI, MD, Charles Stuart	Fairfax
KENNEDY, MD, Mary M	Residents	KNERR, MD, Robt James	Richmond			LEGASPI, MD, Alfredo Lacuna	Fairfax
KENNEDY, MD, Stephen F	Fairfax	KNIGHT JR, MD, Morris Reed	Norfolk			LEGASPI, MD, Amalia G	Chesapeake
KENNEL, MD, Elmer Elwood	Rockingham	KNIGHT, MD, Frank Sutton	Richmond			LEGETT, MD, John Albert	Augusta
KENNEWEG, MD, Donald John	Fredericksburg	KNIGHT, MD, James Gregory	Albemarle			LEGG, MD, Quentin J	Newport News
KENSELL, MD, Ralph F	Rockingham	KNIGHT, MD, Yvonne	Rockingham			LEGIER, MD, Jacques Frederick	Newport News
KENT, MD, James Carl	Prince William	KNISS, MD, Mark Allan	Rockingham			LEGROW, MD, Wynne V E	Southside Va
KENT, MD, James P	Lynchburg	KNOPP, MD, Reuben De Loach	Roanoke			LEGUM, MD, Larry L	Chesapeake
KENT, MD, Richard Irwin	Mid-Tidewater	KNORR, MD, Norman J	Albemarle			LEHMAN, MD, Robt F	Prince William
KENYON JR, MD, Robert Earl	Out Of State	KNOX, MD, Henry Donald	Fairfax			LEHMANN III, MD, Ilse	Richmond
KERMAN, MD, Shelly Lynn	Fairfax	KNUDSON, MD, Homer Ellsworth	Fairfax			LEIDELMEYER, MD, Reinald	Fairfax
KERN, MD, Marguerite Ann	Richmond	KOEHL, MD, Gao Wm	Norfolk			LEMESHEWSKY, MD, Geo P	Alexandria
KERNODLE, MD, Judith M	Richmond	KOEHLER, MD, Rolf Alfred	Fairfax			LENETT, MD, Stephen David	Richmond
KERNODLE, MD, Wm Dwight	Richmond	KOH, MD, Hae Kyung	Hampton			LENGUA, MD, Jose Antonio	Mid-Tidewater
KERNS, MD, John William	Northern Va	KOH, MD, Woon Hi	Hampton			LENTZ, MD, Edmund T	Portsmouth
KERPELMAN, MD, Earle Jerome	Norfolk	KOHLER, MD, Stewart Edwin	Fredericksburg			LEON, MD, Antonio Enrique	Fairfax
KERSEY JR, MD, Wm Ward	Out Of State	KOHN, MD, Gary Mershell	Arlington			LEONCIO, MD, Jose D	Portsmouth
KERSH, MD, Charles R	Residents	KOHRING, MD, Regis Clerke	Rockingham			LEONE, MD, Louis August	Out Of State
KESHMIRI, MD, Abolhasan	Prince William	KOLANSKY, MD, Saul Kalman	Alexandria			LEONHARDT, MD, Hannah V	Out Of State
KESLER, MD, Richard Wm	Albemarle	KOLIA, MD, Gulam-Mohmed M	Arlington			LESKO, MD, Edmund Michael	Roanoke
KESLER, MD, Robt Milton	Norfolk	KOLLER, MD, Miriam K	Residents			LESOWITZ, MD, Sidney Allan	Fairfax
KESSLER II, MD, A. Reif	Roanoke	KOLLER, MD, Stephen Rice	Richmond			LESSIN, MD, Bruce Edward	Fairfax
KESSLER, MD, Geo H	Northern Va	KOLVEREID, MD, Edward Ronald	Fairfax			LEVERTY, MD, Alexander P	Richmond
KESSLER, MD, Carl Paul	Fairfax	KONERDING, MD, Hazel S	Richmond			LEVY, MD, Myron S	Roanoke
KESSLER, MD, Charles A	Norfolk	KONERDING, MD, Karsten F	Richmond			LEVIN, MD, Michael G	Albemarle
KESSLER, MD, Chester Wm	Fairfax	KONTOS, MD, Hermes Apostolou	Richmond			LEVIN, MD, Gershon J	Norfolk
KESSLER, MD, Wm A	Wise	KOONS, MD, Gregory Mark	Fairfax			LEVIN, MD, Stephen M	Alexandria
KETRON, MD, Sami Glimer	Southwest Va	KOONTZ, MD, Warren Woodson	Richmond			LEVINE, MD, Jay Michael	Richmond
KEUTER, MD, Juan Rene	Out Of State	KOPLIN, MD, Julian Arthur	Danville			LEVINE, MD, Leonard S	Fairfax
KEVORKIAN, MD, Constance	Tri-County	KOPP, MD, James Emidio	Norfolk			LEVINE, MD, Richard A	Residents
KEY JR, MD, Wendell Wayne	Richmond	KORKOSZ, MD, Tanya J	Alexandria			LEVINSOHN, MD, Harold Jay	Richmond
KEYS, MD, David Nison	Roanoke	KORN, MD, Robert S	Arlington			LEVITT, MD, Lynn C	Residents
KHACHIKIAN, MD, Gngor	Alexandria	KORNBLUTH, MD, Ralph Ross	Fairfax			LEVY, MD, Donald L	Norfolk
KHAN, MD, Mohammad Aqiq	Fairfax	KORNETSKY, MD, Kenneth M	Fauquier			LEVY, MD, Donald Marvin	Norfolk
KHOMAMI-RAMSEY, MD, Ali	Alexandria	KORNHAUSER, MD, Michael J	Fairfax			LEVY, MD, Edward David	Norfolk
KIBBE, MD, Milton H	Southwest Va	KOSLOW, MD, Joel Lester	Alexandria			LEVY, MD, Jan Alfred	Southwest Va
KICZALES, MD, Adolphe Chas.	Northern Va	KOSTINAS, MD, John E	Portsmouth			LEVY, MD, Philip Morton	Newport News
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KIESEL, MD, Robert D	Arlington	KOTSSELAS, MD, Evangelos N	Prince William			LEWIS, MD, Augustine W	Mid-Tidewater
KIESSLING, MD, Alice H	Fairfax	KOULZAKIS, MD, E N	Fairfax			LEWIS, MD, B. Franklin	Northern Va
KIGHT, MD, John Randolph	Norfolk	KOVAC JR, MD, Michael John	Albemarle			LEWIS, MD, Cyrus Patrick	Southside Va
KILBY, MD, Walter B	Culpeper	KOWLER, MD, Daniel Edward	Richmond			LEWIS, MD, David Howe	Patrick-Henry
KILFEATHER, MD, John E	Fairfax	KOZIOL, MD, Dennis Frank	Virginia Beach			LEWIS, MD, Kerry Randall	Fairfax
KILUK, MD, Kenneth Ignatius	Richmond	KOZIOL, MD, Isaac	Richmond			LEWIS, MD, Richard A	Norfolk
KIM JR, MD, Jin Tek	Richmond	KRAMER, MD, Lloyd Irvin	Fairfax			LEWIS, MD, Richard Gordon	Richmond
KIM, MD, Chin Moon	Prince William	KRAMER, MD, Marc Stephen	Southside Va			LEWIS, MD, Steven T	Franklin
KIM, MD, Chungkook	Fairfax	KRAMER, MD, William	Richmond			LEWIS, MD, Thomas Cary	Rockbridge
KIM, MD, Heeshin	Prince William	KRAUS, MD, Harry Lee	Newport News			LI, MD, Si-Ju	Prince William
KIM, MD, Hie Chul	Arlington	KRAUS, MD, Shane James	Richmond			LIBBY, MD, Russell C	Fairfax
KIM, MD, Hyung Rin	Out Of State	KRAVETZ, MD, Robt Alan	Fredericksburg			LICAMELE, MD, William Louis	Arlington
KIM, MD, Jung-Ah Christina	Albemarle	KREBS, MD, Hans-Bartold	Richmond			LICATA, MD, Robert M	Fairfax
KIM, MD, Kii Seong	Tri-County	KREGER, MD, David Lawrence	Norfolk			LICHTMANN, MD, Albert Leszlo	Arlington
KIM, MD, Mi Yong	Fairfax	KREIDER, MD, Stanley J	Virginia Beach			LIDMAN, MD, Roger W	Norfolk
KIM, MD, Myung Woong	Hampton	KREISLER, MD, Leslie S	Richmond			LIEBERMAN, MD, Michael David	Alexandria
KIM, MD, Sung Yong	Portsmouth	KRELL, MD, Linda Sue	Roanoke			LIEN, MD, Bui Thu	Norfolk
KIM, MD, Young U	Roanoke	KRENTZKY, MD, Stephen Marc	Prince William			LIGHTBURN, MD, Alize Cole	Out Of State
KIMBROUGH, MD, Janet Coleman	Williamsburg	KRESS JR, MD, Sheldon	Fairfax			LILLY JR, MD, Paul Howard	Lynchburg
KIMBROUGH, MD, Raymond D	Fairfax	KRETTEK, MD, John E	Richmond			LILLY, MD, Edward Lewis	Norfolk
KINDRED, MD, Robert G	Out Of State	KRETZ, MD, Wieman H	Newport News			LIM, MD, Angelita Augustin	Norfolk
KING JR, MD, Robert Garland	Richmond	KRICHOVIC, MD, Milan Piersol	Out Of State			LIMAYE, MD, Nirmala S	Arlington
KING, MD, Donald Perry	Richmond	KRISCHER, MD, Meyer I	Richmond			LIN, MD, Hsing-Wu	Southside Va
KING, MD, Elmer Richard	Richmond	KRISHNAMURTHY, MD, Kalale S	Norfolk			LIN, MD, James Min	Richmond
KING, MD, James P	Southwest Va	KROLL, MD, Ronald Neil	Southside Va			LIND, MD, James F	Norfolk
KING, MD, James Peter	Southwest Va	KRON, MD, Irving Louis	Richmond			LINDE, MD, Richard Emil	Fairfax
KING, MD, John Norman	Norfolk	KRONFOL, MD, Nouhad O	Albemarle			LINDEMANN, MD, Lillian C	Richmond
KING, MD, John Winston	Norfolk	KROP, MD, Paul Nicholas	Richmond			LINDOUST, MD, Leo A	Danville
KING, MD, Joseph Willett	Richmond	KROP, MD, Thomas Monroe	Virginia Beach			LINDSAY, MD, Richard Waller	Albemarle
KING, MD, Kenneth R	Newport News	KRUEGER, MD, John Jay	Virginia Beach			LINDSTEDT, MD, Jan Gustaf	Fairfax
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KING, MD, Ronald Lester	Arlington	KUEHN, MD, Hubert W	Residents			LINN, MD, James John	Lynchburg
KING, MD, Wm Whitman	Portsmouth	KUHN, MD, Robt Anthony	Chesapeake			LIPMAN, MD, Ansel	Portsmouth
KINGREE, MD, Wm Blane	Southwest Va	KUIKEN, MD, Garry H	Danville			LIPPARD, MD, Cerroll H	Richmond
KINTIGH, MD, James Wm	Northern Va	KUKICH, MD, Stanka	Floyd			LIPPER, MD, Maurice H	Richmond
KINGZIE IV, MD, Daniel H	Newport News	KULUND, MD, Daniel Nicholas	Fairfax			LIPPERT, MD, John Charles	Danville
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KIRBY, MD, David Alan	Richmond	KUNDRAT, MD, Andrew G	Norfolk			LISZKA, MD, Victor L	Arlington
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KIRKLAND JR, MD, Nathaniel C	Portsmouth	KUPERMINE, MD, Denis S	Richmond			LITTLEPAGE, MD, Eleanor G M	Norfolk
KIRKLAND JR, MD, James A	Albemarle	KURTZKE, MD, John Francis	Richmond			LITTLETON JR, MD, Frederick	Northern Neck
KIRKLAND, MD, Richard Horace	Southside Va	KURUP, MD, Manikoth G	Fairfax			LITTLETON, MD, Philip Ray	Fairfax
	Richmond		Richmond			LITTON, MD, Darlene E B	Wise



LITTON, MD, Frederick Mitchell	Wise	MACIULLA, MD, Louis J	Arlington	MARTIN JR, MD, George W.	Richmond	MC DANIEL, MD, Leroy S	Richmond
LITWILLER, MD, Roger Wayne	Roanoke	MACK, MD, Joseph A.	Tazewell	MARTIN, MD, Arthur J	Fredericksburg	MC DANIEL, MD, Sami M	Norfolk
LIVINGOOD, MD, J.K.	Fairfax	MACK, MD, Theodore R	Richmond	MARTIN, MD, Carolyn Jean	Out Of State	MC DANIELS, MD, L B	Newport News
LIVINGSTON, MD, Stanton K	Out Of State	MACKER, MD, Susan E	Albemarle	MARTIN, MD, Dean H	Fairfax	MC DANNALD JR., MD, Eugene R	Roanoke
LLANERAS, MD, Rene F	Fairfax	MACKINTOSH, MD, Alan	Fairfax	MARTIN, MD, Diana	Fairfax	MC DONALD, MD, David B	Arlington
LLEDO, MD, Alfonso M	Albemarle	MACKLER, MD, Stuart F	Southwest Va	MARTIN, MD, Gerry David	Augusta	MC DONALD, MD, Robert M.	Buckingham
LLEWELLYN, MD, Christine H.	Residents	MACMANUS, MD, Cuentin	Fairfax	MARTIN, MD, John Albert	Roanoke	MC DONALD, MD, Thos D	Buchanan
LLOYD JR., MD, Thos Stacy	Fredericksburg	MACMILLAN, MD, Ralph Victor	Richmond	MARTIN, MD, John Oliver	Fairfax	MC DONOUGH, MD, Wm Wallace	Richmond
LLOYD JR., MD, Samuel J	Fauquier	MACON, MD, Edward Malcolm	Out Of State	MARTIN, MD, Lee B	Arlington	MC DOWALL, MD, Jos T	Fairfax
LLOYD, MD, Wm S	Louisa	MACYS, MD, Joseph R	Richmond	MARTIN, MD, Moir Glenwood	Southwest Va	MC DOWELL, MD, Alice W	Southwest Va
LOAR, MD, Charles Richard	Out Of State	MADDOX, MD, Joseph Edward	Northern Va	MARTIN, MD, Randolph P	Albemarle	MC DOWELL, MD, Charles L	Richmond
LOCKHART, MD, John Lee	Newport News	MADGE, MD, Gordon Evans	Richmond	MARTIN, MD, Shirley S	Arlington	MC EENTE, MD, James Phillip	Richmond
LODERSTEDT, MD, Gunther J	Southwest Va	MADONIA, MD, Eugene C	Patrick-Henry	MARTIN, MD, Wm Leroy	Virginia Beach	MC EENTE, MD, Wesley Edward	Richmond
LOESCH, MD, Beverly Jean	Augusta	MAFFEY, MD, Ralph B	Out Of State	MARTIN, MD, Wm Watkins	Richmond	MC FADEN, MD, Jos T	Norfolk
LOEW, MD, Albert G	Portsmouth	MAGANIAS, MD, Nicholas H	Fairfax	MARTINELLI, MD, Maurice Ivan	Virginia Beach	MC FADDIN, MD, James G	Out Of State
LOFTIN III, MD, Chas Ivey	Roanoke	MAGEE JR., MD, William P	Norfolk	MARTINEZ, MD, Horacio Duarte	Albemarle	MC FARLAND, MD, John W	Fairfax
LOGANATHAN, MD, Sri Tharan	Richmond	MAGEE, MD, Archibald C.	Out Of State	MARTIROSIAN, MD, Edward D	Richmond	MC GAVIN, MD, Thos A	Arlington
LOHR, MD, Jacob Andrew	Albemarle	MAGIER, MD, Igor	Chesapeake	MARVEN, MD, Lee Jonathan	Virginia Beach	MC GEE JR., MD, James E	Out Of State
LOIACONO, MD, Patsy Julius	Hampton	MAGIER, MD, Nina G	Roanoke	MASCARINAS JR., MD, Teofilo C	Virginia Beach	MC GHEE JR., MD, Read F	Richmond
LONERGAN, MD, Maryrose T.	Residents	MAGNANT, MD, Geo Arthur	Fairfax	MASICA, MD, Daniel N	Richmond	MC GHEE, MD, Judith F	Richmond
LONG JR., MD, Alvin Penrose	Portsmouth	MAGNESS II, MD, Aitred P.	Norfolk	MASLOFF, MD, James Irvin	Albemarle	MC GINN, MD, James Sylvester	Patrick-Henry
LONG JR., MD, John A	Arlington	MAGPOC, MD, Norma	Virginia Beach	MASON JR., MD, James D	Southside Va	MC GOUGH, MD, Thos F	Alexandria
LONG, MD, Albert Emanuel	Alexandria	MAGRUDER, MD, R. Gregory	Albemarle	MASON, MD, Gordon L.	Tri-County	MC GOVERN, MD, Francis H	Danville
LONG, MD, George E.	Arlington	MAHAN, MD, Jack Delano	Norfolk	MASON, MD, Joel A	Virginia Beach	MC GUIRE JR., MD, Hunter H	Richmond
LONG, MD, James Arthur	Augusta	MAHINI III, MD, Abraham	Tri-County	MASON, MD, Mark S.	Norfolk	MC GUIRE, MD, Erma J Marra	Southwest Va
LONGACHER JR., MD, Jos Wm	Richmond	MAHINPOUR, MD, Siavash	Alexandria	MASRI, MD, Asad M	Prince William	MC GUIRE, MD, Lockhart Bemiss	Albemarle
LONGAN JR., MD, Robt C	Richmond	MAHNESMIT, MD, Randolph C.	Augusta	MASRI, MD, Faig Asad	Southside Va	MC ILWAINE III, MD, Wm B	Southside Va
LONGFORD, MD, Desmond	Tri-County	MAILLIS, MD, Maxwell Sherwood	Fairfax	MASRI, MD, Louis Benedict	Tazewell	MC INTYRE, MD, William Wallace	Northampton
LONGNECKER, MD, David E	Albemarle	MAIZELS, MD, Max Sam	Wise	MASSAD, MD, Thos A	Fredericksburg	MC KAIN, MD, Carey W.	Southwest Va
LONGO, MD, Antonio Miguel	Alexandria	MAJEWSKI, MD, Allen David	Richmond	MASRO, MD, Thos A	Albemarle	MC KAY, MD, James	Richmond
LOONEY, MD, Wm Boyd	Albemarle	MAJESSI, MD, Heshmatolah	Lynchburg	MASSEY III, MD, William Jos	Fredericksburg	MC KEOWN, MD, Chas E	Richmond
LOPEZ, MD, Rodolfo L.	Arlington	MAJOR, MD, Mary Jane	Rockingham	MASSEY JR., MD, John Wm	Williamsburg	MC KIBBIN, MD, Douglas W	Augusta
LOPEZ-TOCA, MD, Ruben	Alexandria	MAKAROWSKY, MD, Eugene	Fairfax	MASSEY JR., MD, Chas Webster	Newport News	MC KNELLY, MD, Larry Oren	Alexandria
LORDI, MD, William M	Richmond	MALCHOFF, MD, Carl D.	Richmond	MASSEY, MD, Philip N.	Richmond	MC LAUGHLIN, MD, Robert E	Albemarle
LORENZ, MD, Martin Fred	Virginia Beach	MALCOLM, MD, Bradley Scott	Northern Va	MASIE, MD, F Standford	Richmond	MC LEAN, MD, Walter C	Albemarle
LORENZ, MD, Richard Lawrence	Prince William	MALDONADO, MD, Luis Gonzalo	Richmond	MASIE, MD, Saml Powell	Stuart	MC LEOD, MD, Harry Ronald	Loudoun
LORIMER, MD, William V.	Danville	MALEK, MD, Nabil Shehata Rizk	Out Of State	MASIE, MD, Wm Mc Kinnon	Lynchburg	MC MAHON, MD, Geo Jos	Arlington
LORIO, MD, Jos Philibert	Fairfax	MALIN, MD, Wendell Eugene	Out Of State	MASTERSON, MD, James H	Fairfax	MC MANUS, MD, Reginald Paul	Arlington
LOTANO, MD, Remo Andrea	Lynchburg	MALKA, MD, Jeffrey S	Southwest Va	MASTROTA, MD, Francis M	Fairfax	MC MASTER, MD, Delphine A	Arlington
LOTZ, MD, Myron	Fairfax	MALLARE, MD, Melchor Pulido	Fairfax	MATCHETT, MD, Robert M	Virginia Beach	MC MULLAN, MD, Francis H	Richmond
LOU, MD, Ek Seng	Prince William	MALLIET, MD, Jennifer L.	Danville	MATTHEWS JR., MD, J Lee	Prince William	MC MURRER JR., MD, James P	Fairfax
LOUGHEED, MD, Marvin N	Roanoke	MALNETT, MD, Jonathan K.	Residents	MATTHEWS, MD, Emmett C	Richmond	MC NAMEE JR., MD, Edwin T	Stuart
LOUGHRIDGE, MD, Chalmers A	Alexandria	MALONEY, MD, Wm F	Rockingham	MATTHEWS, MD, John Addison	Lynchburg	MC NEELY, MD, Irwin Hollar	Tri-County
LOUKA, MD, Kamal S	Residents	MALPANI, MD, Kalidas D.	Out Of State	MATHIAS, MD, Jos E	Arlington	MC NEER, MD, Paul Randolph	Richmond
LOUW, MD, Jan Cornelius	Norfolk	MALTA, MD, Vito J	Northern Va	MATSON, MD, Raymond Eugene	Lynchburg	MC NEILL, MD, Donald Hanson	Northern Va
LOVE, MD, Carolyn A	Virginia Beach	MALTESE, MD, Frances Anna	Richmond	MATSHUSHIGE, MD, Kouichi	Fredericksburg	MC QUEEN, MD, Robert C	Northern Va
LOVE, MD, Suzanne S	Richmond	MAMANA, MD, John Philip	Fairfax	MATTEW, DO, John O A	Portsmouth	MC RAE, MD, Marvin E	Out Of State
LOVELL JR., MD, Charles F	Norfolk	MANALO JR., MD, Buenaventura	Southwest Va	MATTHEWS, MD, Richard Eugene	Newport News	MC WHORTER, MD, W David	Northern Va
LOVINGER, MD, Robert D	Richmond	MANALO, MD, Bayani L.	Fairfax	MATTHEWS, MD, Robt Geo	Richmond	MC WILLIAMS, MD, Thos G	Arlington
LOW, MD, James R	Fredericksburg	MANCUSO, MD, Frank Smith	Fairfax	MAUCK JR., MD, Henry P	Fairfax	MCCABE, MD, Dennis J	Fairfax
LOWE JR., MD, Richard H	Roanoke	MANDANIS, MD, John P	Richmond	MAUCK, MD, Robert H.	Patrick-Henry	MCCABE, MD, Thomas Ambrose	Arlington
LOWE, MD, Scott Miller	Norfolk	MANDEL, MD, Michael David	Arlington	MAUCK, MD, William R	Richmond	MCCARTHY, MD, Harry Smith	Newport News
LOWELL, MD, William G	Norfolk	MANDELSTAMM, MD, Maria T	Patrick-Henry	MAURONER JR., MD, Norman L	Fauquier	MCCCLURE JR., MD, Claude	Danville
LOWEN, MD, Beal Aptheker	Alexandria	MANDES, MD, Thomas C.	Richmond	MAX, MD, Martin H	Norfolk	MCCCLURE, MD, Phillip H	Southside Va
LOWRY, MD, Mann T	Hanover	MANETZ, MD, Charles E	Fairfax	MAY, MD, Ellis F	Newport News	MCCORMACK, MD, Regina Claire	Albemarle
LOXLEY, MD, Sidney S	Chesapeake	MANGOLD, MD, Harry Armstrong	Richmond	MAY III, MD, James Terrell	Richmond	MCCGEE JR., MD, Francis E	Richmond
LUBLIN, MD, Bernard A	Richmond	MANGUKIAN JR., MD, Dertad	Fairfax	MAY JR., MD, Virgil R	Richmond	MCCGLYNN, MD, Fred J	Richmond
LUCARIC JR., MD, Zdenko	Out Of State	MANGUS, MD, Julian Edward	Richmond	MAY, MD, Dean Francis	Alexandria	MCCGRATH, MD, Francis J	Arlington
LUCAS JR., MD, Kenneth Wilson	Franklin	MANHEIM, MD, Arnold	Danville	MAY, MD, Madge D.	Northampton	MCCGROARTY, MD, David John	Richmond
LUCAS, MD, Davis C	Roanoke	MANICKAVASAGAR, MD, Marie J	Fairfax	MAY, MD, Russell Leon	Fairfax	MCCILWAINE, MD, Benjamin H.	Southside Va
LUCAS, MD, Thos L	Out Of State	MANICKAVASAGAR, MD, S.	Norfolk	MAY, MD, William H	Norfolk	MCCLEOD, MD, James William	Mid-Tidewater
LUCCOLI JR., MD, Lucio	Alexandria	MANLAPAZ, MD, Carolina P	Fairfax	MAYER, MD, Andrew Anthony	Norfolk	MCLINTOCK, MD, M. Gillian	Albemarle
LUCEY, MD, John D.	Fredericksburg	MANLEY JR., MD, Walter F	Richmond	MAYER, MD, Walter	Richmond	MCCNEER, MD, Keith Wilson	Richmond
LUCKY, MD, James Thos	Southwest Va	MANLEY, MD, Walter F	Roanoke	MAYER, MD, William Dixon	Norfolk	MCTAMANEY, MD, James Paul	Richmond
LUDEMAN JR., MD, Douglas H.	Residents	MANN, MD, Dean Le Mar	Roanoke	MAYERS JR., MD, Stanley P	Out Of State	MEADE JR., MD, Thomas S	Virginia Beach
LUEDKE, MD, George Wm	Roanoke	MANN, MD, Geoffrey T	Out Of State	MAYES, MD, Kenneth Lee	Portsmouth	MEADOR, MD, Blake W	Richmond
LUKE, MD, Mary Jane	Allegheny	MANN, MD, H. Thompson	Portsmouth	MAYO JR., MD, Lemuel E	Portsmouth	MEDFORD, MD, Frank Eldridge	Newport News
LUKOWSKY, MD, Gerhard Hans	Alexandria	MANN, MD, James Packard	Richmond	MAYO, MD, Fitzhugh	Richmond	MEDSKER, MD, Thos M.	Fredericksburg
LUM, MD, Natalie Jean	Southside Va	MANN, MD, Robt Fletcher	Alexandria	MAYSON JR., MD, Preston B	Roanoke	MEES, MD, Charles H	Richmond
LUMPKIN, MD, Martha Ray	Arlington	MANNING, MD, George S.	Norfolk	MAZUR, MD, Stephen H	Williamsburg	MEES, MD, Thos George	Richmond
LUNA, MD, Federico Martin	Norfolk	MANNING, MD, Preston Cocke	Augusta	MC ADAM, MD, Richard Bernard	Hampton	MEHRHOF JR., MD, Austin I.	Richmond
LUNA, MD, Ruben Villaflores	Fredericksburg	MANOHARAN, MD, E	Wise	MC ALLISTER SR., MD, Russell	Richmond	MEIGHAN, MD, Michael M.	Residents
LUNDEEN, MD, Wm Bruce	Arlington	MANSHFIELD, MD, John Bristow	Northern Va	MC ALLISTER, MD, John Eldon	Northern Va	MEILLER, MD, Joan Mason	Richmond
LUNDIE, MD, Donald Wayne	Tri-County	MANSON, MD, R Campbell	Norfolk	MC ALPINE, MD, Robt E	Norfolk	MEINCKE, MD, David Lee	Southwest Va
LUSTIG, MD, David M	Virginia Beach	MANWARING, MD, John L	Richmond	MC ATEER, MD, Gerald H	Fairfax	MEISTER, MD, Robert Jay	Arlington
LUTH, MD, Janice Elaine	Bedford	MAOURY, MD, Stanley D	Fauquier	MC AVENEY, MD, William J	Fairfax	MELCHIONNA, MD, Olin Richard	Roanoke
LUTHER, MD, Burdon Lowe	Roanoke	MAPHIS JR., MD, Frederick D	Prince William	MC BRAYER JR., MD, Reuben H	Norfolk	MELLA, MD, Barbara A	Fairfax
LUTHRA, MD, Ramesh C	Wise	MAPP, MD, John R	Wise	MC CABB, MD, Wm Otey	Lynchburg	MELLETT, MD, M Susan J	Richmond
LUTTRELL, MD, Homer B	Southwest Va	MAPP, MD, John R	Virginia Beach	MC CAHILL, MD, Thos D	Richmond	MELMED, MD, Allan Stanley	Fairfax
LUTZ, MD, Roy Winston	Northern Va	MARAK JR., MD, Geo Edward	Northampton	MC CANN, MD, Wm John	Fairfax	MELNICK, MD, Irving	Danville
LUX, MD, Ann Mary	Fairfax	MARCUS, MD, Norman A.	Alexandria	MC CARTHY, MD, William C.	Prince William	MELONI, MD, Chas Robt	Alexandria
LYLE, MD, John P.	Tri-County	MARSH, MD, Carey Miles	Fairfax	MC CARTY, MD, Dennis P.	Northern Va	MELTON III, MD, John Wesley	Out Of State
LYLE, MD, Lurton Braxton	Buchanan	MARSHALL JR., MD, Jos K	Norfolk	MC CAUSLAND, MD, Alexander	Roanoke	MELTON, MD, Harvey Edward	Northern Va
LYLES JR., MD, John Wm	Fairfax	MARSHALL JR., MD, Hubert A.	Fairfax	MC CLANE, MD, John Raymond	Southwest Va	MELZIG, MD, Eric Perry	Richmond
LYNCH II., MD, Vernon Lee	Franklin	MARSHALL, MD, Charles B.	Northern Va	MC CLELLAN, MD, Jason E	Newport News	MEZDEZ, MD, Hedley Norman	Hampton
LYNCH JR., MD, Donald F.	Danville	MARSHALL, MD, Douglas Lyle	Fairfax	MC CLOUD, MD, Deborah J	Patrick-Henry	MEZDEZ, MD, Manuel Diaz	Richmond
LYNCH, MD, George Michael	Fairfax	MARSHALL, MD, John Lyons	Tazewell	MC CLUNEY, MD, Kerry W	Rockbridge	MENDEZ III, MD, Renaldo	Southside Va
LYNCH, MD, James M.	Arlington	MARSHALL, MD, Lawrence Vinton	Rockbridge	MC CLUNG JR., MD, Oscar H	Fairfax	MENK, MD, Karl F	Augusta
LYNCH, MD, John P	Richmond	MARSLAND, MD, David Wilson	Fairfax	MC CLURE, MD, Wm West	Rockbridge	MENSCH, MD, Arthur H	Fairfax
LYONS JR., MD, Sidney	Arlington	MARTELS JR., MD, Leon Alphonse	Richmond	MC COLLUM, MD, Donald C	Out Of State	MENZER JR., MD, Robert M	Albemarle
		MARKHAM, MD, Harold Wm	Fairfax	MC CONAHEY III, MD, Wm M	Halifax	MERCER, MD, H. Richard	Richmond
		MARKHAM, MD, J. David	Culpeper	MC CORKE, MD, Robt Leroy	Mid-Tidewater	MERCHANT III, MD, Wilson C.	Richmond
		MARKOWITZ JR., MD, Martin	Norfolk	MC CORMICK, MD, Hugh Bernard	Newport News	MEREDITH JR., MD, George Minor	Norfolk
		MARKOWITZ, MD, Michael Paul	Richmond	MC COY, MD, Cullen M	Norfolk	MEREDITH II, MD, H Clarkson	Norfolk
		MARKS, MD, Frank Wayland	Fredericksburg	MC CRAW, MD, Stephen Hartzell	Norfolk	MERKEL, MD, Victoria Lessinger	Residents
		MAROTTO JR., MD, Felix	Norfolk	MC CREADY, MD, Danl Roy	Norfolk	MERKER, MD, Frank F	Southwest Va
		MARSELLA, MD, John Jerry	Danville	MC CUE III, MD, Frank C	Virginia Beach	MERLE-IGNACIO, MD, Eleodora C	Norfolk
		MARSH, MD, John O.	Arlington	MC CUE JR., MD, Howard M	Albemarle	MERLINO, MD, Robin Beth	Arlington
		MARSHALL JR., MD, Jos K	Residents	MC CUE JR., MD, Carolyn Moore	Richmond	MERO, MD, James Hill	Fairfax
		MARSHALL JR., MD, Hubert A.	Albemarle	MC CUNE, MD, Frederick K	Richmond	MERRICK, MD, H. Curtiss	Norfolk
		MARSHALL, MD, Charles B.	Patrick-Henry	MC CUTCHEON JR., MD, R	Virginia Beach	MERRILL, MD, Cynthia Westneat	Richmond
		MARSHALL, MD, Douglas Lyle	Northern Va	MC DAD, MD, John Patrick	Richmond	METZGER, MD, Arthur Zelig	Fairfax
		MARSHALL, MD, John Lyons	Newport News	MC DANIEL JR., MD, Eugene M	Alexandria	MEWBERNE, MD, Edward B	Newport News
		MARSHALL, MD, Lawrence Vinton	Floyd	MC DANIEL JR., MD, David H	Out Of State	MEYER, MD, Julien H	Roanoke
		MARSLAND, MD, David Wilson	Richmond	MC DANIEL, MD, James Lund	Virginia Beach	MEYER, MD, Russel	Virginia Beach
		MARTELS JR., MD, Leon Alphonse	Fairfax	MC DANIEL, MD, John Fredrick	Northampton	MEYERHOFF, MD, Geo Edward	Richmond
		MARTENS JR., MD, Werner	Norfolk	MC DANE, MD, Rene S	Norfolk		Fairfax
		MARTENSON, MD, Stephen H.	Northern Va				
		MARTIN II, MD, Lewis K	Northern Va				
		MARTIN JR., MD, Berkeley H	Richmond				
		MARTIN JR., MD, Lee Baldwin	Arlington				



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REINHARDT, MD, Erich Manfred	Arlington
REINHARDT, MD, Stephen D	Residents
REINH, MD, Wm Edwin	Rockingham
RELYEA, MD, Richard Lee	Out Of State
REMANADABAN, MD, Teodulo	Roanoke
REMUZZI, MD, Robert	Loudoun
RENALDO, MD, Gary Joseph	Residents
RENFIELD, MD, Marilyn Lewis	Fairfax
RENICK, MD, Ole Witholm	Roanoke
RENNIE, MD, Laurie Earl	Richmond
REPASS, MD, James Albert	Virginia Beach
REPASS, MD, James Caldwell	Southwest Va
REPASS, MD, Robt A	Norfolk
RESHEFSKY, MD, Bonnie Louis	Albermarle
RESPESS, MD, James C	Alexandria
RESTIVO, MD, Marion Chas	Northern Va
REULING JR, MD, Frank Harold	Fairfax
REYNOLDS JR, MD, Arthur M	Fairfax
REYNOLDS, MD, Brian Joe	Fredericksburg
REYNOLDS, MD, George A	Richmond
REYNOLDS, MD, Richard R	Culpeper
REYNOLDS, MD, Thomas E	Northern Va
REZBA, MD, Benjamin Victor	Alexandria
RHAME, MD, Richard Coleman	Residents
RHEA, MD, Randall R	Prince William
RHOADS, MD, John Chas	Out Of State
RHODE, MD, Joseph G	Bedford
RHODES JR, MD, Hebert Paul	Northern Va
RHODES, MD, Mark A	Newport News
RHODES, MD, Ray F	Fairfax
RHYMERS, MD, Kurt Lee	Fairfax
RIBEIRO, MD, Gilbert	Newport News
RICCARDO, MD, Luzviminda	Hampton
RICCIARELLI, MD, Giacomo A	Richmond
RICE JR, MD, Marion Lee	Southside Va
RICE, MD, Benjamin Holt	Residents
RICE, MD, Diane Beach	Roanoke
RICE, MD, James Davies	Norfolk
RICE, MD, Marcus Charles	Fairfax
RICH III, MD, William L	Amherst-Neelson
RICH, MD, Elizabeth J	Southside Va
RICHARD, MD, Louis E	Richmond
RICHARDS JR, MD, Chas N	Roanoke
RICHARDS JR, MD, Lewis G	Out Of State
RICHARDS, MD, A Dewey	Alexandria
RICHARDS, MD, Ashby Turner	Richmond
RICHARDS, MD, Nelson G	Northern Va
RICHARDSON, MD, Don Harlor	Albermarle
RICHARDSON, MD, Donald R	Loudoun
RICHARDSON, MD, Douglas S	Richmond
RICHARDSON, MD, Herman M	Augusta
RICHARDSON, MD, Peter Bruce	Patrick-Henry
RICHMAN, MD, Donald Wm	Patrick-Henry
RICHMOND, MD, Marion D	Prince William
RICHTER, MD, Geraldine	Out Of State
RICHTER, MD, Paul	Arlington
RICKERICH, MD, Chas L	Rockingham
RIDDEL JR, MD, Clifford T	Albermarle
RIDDERVOLD, MD, Hans Olav	Chesapeake
RIDDICK JR, MD, Joseph H	Richmond
RIDDICK, MD, David Haydon	Patrick-Henry
RIDER, MD, Robt Edward	Roanoke
RIDGWAY-HULL, MD, Duvahl B	Out Of State
RIEMAN, MD, G Fletcher	Fairfax
RIFAAT, MD, Monira K	Lynchburg
RIGGINS JR, MD, William M	Residents
RIGGLE, MD, Karl P	Lynchburg
RILEY III, MD, Harold Lee	Richmond
RILEY JR, MD, Harold Lee	Northern Va
RILEY, MD, Chas R	Richmond
RILEY, MD, Chester Loris	Richmond
RILEY, MD, P. Thomas	Newport News
RINALDI, MD, Italo Pio	Prince William
RINGLER, MD, John Geo	Southwest Va
RIOS, MD, Juan Francisco	Albermarle
RIPBERGER, MD, Frank M	Roanoke
RIPLEY, MD, Louis Paul	Virginia Beach
RIPOLL, MD, Ignacio	Norfolk
RISH, MD, Berkley Lamont	Lynchburg
RISHER, MD, John Calhoun	Richmond
RITCHIE JR, MD, Geo G	Richmond
RIVADENEIRA, MD, Colon	Richmond
RIVERS, MD, Cullen B	Alexandria
RIXSE, MD, Robt Sheldon	Out Of State
RIZKALLA, MD, Sam N	Alexandria
ROARK, MD, John W	Fairfax
ROATH, MD, Michael Steven	Fredericksburg
ROBBINS III, MD, Clement Jay	Patrick-Henry
ROBBINS JR, MD, Wm Lacy	Southside Va
ROBBINS, MD, Clayton Asa	Norfolk
ROBBINS, MD, Joseph A	Alexandria
ROBBINS, MD, Kenneth Xenophon	Fairfax
ROBECK, MD, Ilene Rae	Out Of State
ROBERT, MD, Dyer	Hampton
ROBERT, MD, Frank Chambers	Norfolk
ROBERTO, MD, Frank A	Residents
ROBERTS II, MD, Allen H	Halifax
ROBERTS JR, MD, Lucien Wood	Newport News
ROBERTS, MD, Bobbie Lee	Richmond
ROBERTS, MD, Bruce Taylor	Hampton
ROBERTS, MD, Ernest S	Alexandria
ROBERTS, MD, John Edmund	Augusta
ROBERTSON JR, MD, Alex F	Richmond
ROBERTSON JR, MD, John Mott	Lynchburg
ROBERTSON JR, MD, Charles H	Richmond
ROBERTSON, MD, Elmer S	Richmond
ROBERTSON, MD, Louise Wilkes	Richmond
ROBERTSON, MD, Robt John	Virginia Beach
ROBERTSON, MD, William A	Richmond

ROBERTSON, MD, Wm Clayton	Portsmouth
ROBESON, MD, Ella P Tompkins	Hampton
ROBINETT, MD, Paul Ward	Portsmouth
ROBINETTE, MD, Emory H.	Southwest Va
ROBINS JR., MD, Spotswood	Richmond
ROBINS, MD, Richard Bailey	Newport News
ROBINSON II., MD, Frederick L	Newport News
ROBINSON III., MD, Grover C	Richmond
ROBINSON JR., MD, Dennis H	Bedford
ROBINSON, MD, Frederick Dani	Richmond
ROBINSON, MD, James F	Richmond
ROBINSON, MD, James P.	Hampton
ROBINSON, MD, Jos Alexander	Tazewell
ROBINSON, MD, Ralph M	Fauquier
ROBINSON, MD, Wm Mayer	Richmond
ROBLETE, MD, Beulah V	Alleghany
ROBSON, MD, Scott M.	Alexandria
ROCHESTER, MD, Dudley F	Albemarle
ROCHWIS, MD, Ann Romatowski	Fairfax
ROCHWIS, MD, Paul Gregor	Fairfax
RODA, MD, Prospero De La Cruz	Mid-Tidewater
RODGERS, MD, Bradley M.	Albemarle
RODGERS, MD, Stephen Ouarles	Virginia Beach
RODGERS, MD, Terry C	Norfolk
RODILOSSO, MD, Philip Thos	Arlington
RODMAN, MD, James M.	Lynchburg
RODRIGUEZ JR., MD, Claudio	Norfolk
RODRIGUEZ, MD, Oscar	Alexandria
ROEBUCK, MD, Basil Enoch	Southwest Va
ROEBUCK, MD, Jerome Barland	Southside Va
ROGERS JR., MD, Henry Moore	Norfolk
ROGERS JR., MD, Richard O	Tri-County
ROGERS, MD, Charles L.	Fairfax
ROGERS, MD, John Fredric	Richmond
ROGERS, MD, William E.	Augusta
ROGERS, MD, Wm Hamilton	Tri-County
ROGNEY, MD, Douglas Lloyd	Southwest Va
ROGOL, MD, Alan David	Albemarle
ROLL, MD, Cornelis	Danville
ROLL JR., MD, William E	Fairfax
ROLLER, MD, Gerald Wm	Roanoke
ROLLINS, MD, Dixon Michael	Tri-County
ROMAINE, MD, Chas Nichols	Richmond
ROMAN, MD, Jorge	Roanoke
ROMANSKY, MD, Stephen Hess	Alexandria
ROMERO, MD, Aiel G	Norfolk
ROMERO, MD, Gonzalo	Fairfax
RONNESS, MD, Joseph O	Arlington
RONOUILLO, MD, Honorio	Out Of State
ROOK, MD, Frederick W	Arlington
ROONEY, DO, Dan Dare	Fairfax
ROONEY, MD, Mervyn Stuart C	Richmond
ROOT, MD, Carl Fredrick	Portsmouth
ROPER, MD, Albert L	Norfolk
ROPER, MD, Barry Edward	Richmond
ROSAN, DO, Stuart Wm	Out Of State
ROSANELLI JR., MD, Peter	Richmond
ROSE JR., MD, John B	Fredericksburg
ROSE JR., MD, Leslie Wm	Richmond
ROSE, MD, Earl F	Out Of State
ROSEBLAT, MD, Aldo M.	Arlington
ROSEN, MD, David Irving	Out Of State
ROSEN, MD, Leonard A	Fairfax
ROSENBAUM JR., MD, Meyer	Arlington
ROSENBAUM, MD, Geo R	Out Of State
ROSENBERG, MD, Leon Harris	Out Of State
ROSENBERG, MD, Maurice S.	Williamsburg
ROSENBERG, MD, Sanford M	Richmond
ROSENFELD, MD, Stephen P	Alexandria
ROSENOFF, MD, Stephen Howard	Roanoke
ROSENTHAL, MD, James	Residents
ROSENTHAL, MD, Macey H	Lynchburg
ROSENTHAL, MD, Richard R	Fairfax
ROSENTHAL, MD, Sheldon Jay	Alexandria
ROSENTHAL, MD, Steve	Fairfax
ROSS JR., MD, David Aaron	Portsmouth
ROSS JR., MD, Wm Tyler	Albemarle
ROSS, MD, Michael A.	Fairfax
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ROSSHEIM, MD, Edgar Herbert	Norfolk
ROSSI, MD, Gustavo A	Arlington
ROSSILLO, MD, Ludwig Anthony	Out Of State
ROTCHFORD, MD, James Patrick	Arlington
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ROTH, MD, Robt Frank	Roanoke
ROTHMAN, MD, Atan Lee	Residents
ROUDAY, MD, William A	Fairfax
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ROUTSON, MD, Gary Wayne	Patrick-Henry
ROWE, MD, Douglas Stephen	Richmond
ROWE, MD, Henry C.	Mid-Tidewater
ROWELL, MD, Frank E	Norfolk
ROWLAND JR., MD, Harry S	Richmond
ROWLAND JR., MD, Robert C.	Chesapeake
ROWLINGSON, MD, John Clyde	Albemarle
ROY, MD, Gaston E	Fairfax
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ROYAL, MD, Harry Willis	Richmond
ROYCROFT, MD, David Wm	Patrick-Henry
ROYER, MD, Thos C	Norfolk
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ROYSTER, MD, Henry Page	Richmond
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RUDZINSKI, MD, Dennis J	Richmond
RUFFIN JR., MD, Willcox	Norfolk
RUFFIN, MD, Herbert G	Richmond
RUHNKE JR, MD, Edward E	Southside Va
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RUIZ, MD, Abelardo Antonio	Portsmouth
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RUONA, MD, Luanne	Alexandria
RUSHER, MD, Wm De Witt	Richmond
RUSHIA, MD, Edwin Le	Albemarle
RUSHIA, MD, Mary Anna	Albemarle
RUSS JR, MD, Clerke	Virginia Beach
RUSSELL JR, MD, John A	Richmond
RUSSELL, MD, Arch S	Mid-Tidewater
RUSSI, MD, Simon	Southside Va
RUSSO, MD, Eugene P	Fairfax
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RUTH, MD, Gerald Jay	Roanoke
RUTHERFORD JR, MD, William	Roanoke
RUTKOWSKI, MD, Robert	Culpeper
RYAN, MD, Arthur Eugene	Richmond
RYAN, MD, John Edward	Arlington
RYAN, MD, John Thomas	Fredericksburg
RYAN, MD, Mary C	Fairfax
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RYAN, MD, Robert Francis	Arlington
RYAN, MD, Thomas J	Arlington
RYDER, MD, Craig A	Tri-County
RYPLANSKY, MD, Anatol	Southwest Va
RYU, MD, Jai Yoi	Alexandria
S	
SABELLA, MD, Donald A	Fairfax
SABELLA, MD, Lereine	Fairfax
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SACKETT, MD, Chas H	Lynchburg
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SACKS, MD, Eugene Ira	Augusta
SADIGHIAN, MD, Z Dean	Southside Va
SADAJADI III, MD, Parviz-Mohsen	Allegheny
SADLER JR, MD, Charles R	Virginia Beach
SADLER, MD, Wm Anderson	Mid-Tidewater
SADOVE, MD, Richard C	Residents
SADR, MD, Manijeh	Fairfax
SAENZ, MD, Enrique Antonio	Norfolk
SAGER, MD, Allen Robt	Fairfax
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SCOTT JR., MD, David Wm	Fredericksburg
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SCOURAS, MD, Geo Pete	Patrick-Henry
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SCUTERO, MD, James Vincent	Portsmouth
SEAL, MD, R.B.	Portsmouth
SEALE, MD, Danl Logan	Albemarle
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SEASE, MD, James Richard	Rockingham
SEASE, MD, Robt H	Rockingham
SEBASTIAN, MD, Jos A	Fairfax
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SEEHERRMAN, MD, Robt S	Virginia Beach
SEEMAN, MD, Irvin Jay	Richmond
SEGALL, MD, Errol Alan	Fairfax
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SEIBERT, MD, John Douglas	Chesapeake
SEIF III, MD, Rahmat	Roanoke
SEILER JR., MD, Ira	Fairfax



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SEITZ, MD, Donald G	Richmond	SIEGEL, MD, Fred H	Chesapeake	SMITH, MD, Stuart James	Hampton	STANFORD JR, MD, Sam R	Mid-Tidewater
SELTZ, MD, Georg Karl	Prince William	SIEVERT, MD, Jose L	Out Of State	SMITH, MD, Thomas Adrian	Richmond	STANFORD, MD, Walter J	Southwest Va
SELDEN, MD, Robt Francis	Albemarle	SIEWICK, MD, Jos W	Fairfax	SMITH, MD, Thomas Henry	Southwest Va	STANTON, MD, Archie C	Newport News
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SELIG JR, MD, Julian Wood	Out Of State	SILBER, MD, Gershon	Roanoke	SMITH, MD, Wm Crenshaw	Augusta	STARK, MD, James J	Norfolk
SELLERS, MD, John G	Norfolk	SILBERMAN, MD, Edward K	Alexandria	SMOKVINA, MD, Drago	Fairfax	STARKMAN, MD, Martin T	Richmond
SELLMAN, MD, James E	Richmond	SILBERSIEPE, MD, Heinz-Otto	Fauquier	SMOKVINA, MD, Marija Demsar	Fairfax	STARLING JR, MD, James F	Danville
SELPH, MD, John William	Patrick-Henry	SILEO, MD, Robert Peter	Arlington	SMOOT, MD, John Lewis	Fredericksburg	STARR, MD, John Walter	Roanoke
SEMPCHYSHYN, MD, Geo O	Richmond	SILVERMAN, MD, Herbert R	Danville	SNEAD IV, MD, John Peyton	Culpeper	STATHOS JR, MD, John A	Augusta
SEMENDY, MD, Valeri P	Fairfax	SILVERMAN, MD, Joel J	Richmond	SNEAD, MD, Lawrence O	Richmond	STAUFFER, MD, John M	Rockingham
SEMMES, MD, Benedict J	Alexandria	SILVESTER, MD, Michael Joseph	Orange	SNEAD, MD, James Given	Northern Va	STAVOLA, MD, Anthony R	Roanoke
SENATORE, MD, Peter J	Residents	SILVESTER, MD, Timothy James	Lynchburg	SNEAD, MD, Ronald Wilson	Richmond	STAY, MD, Ellsworth J	Arlington
SENDI, MD, Houchang	Alexandria	SIM, MD, Peter Alan	Williamsburg	SNEAD, MD, Russell N	James River	STEC, MD, Rita Jeanne	Residents
SENECA, MD, Russell P	Fairfax	SIMANIS, MD, Juris	Alleghany	SNELL, MD, Henry Madison	Out Of State	STECKER JR, MD, John F	Norfolk
SENFIELD, MD, Richard Maxon	Richmond	SIMMONS, MD, Robt Geo	Danville	SNIDER, MD, Gary Boyd	Norfolk	STECKLER, MD, Eric Alan	Fairfax
SENNESH, MD, Joel David	Fairfax	SIMON, MD, Robert Isaac	Arlington	SNIDER, MD, Gilbert M	Norfolk	STEELE, MD, Chas W	Tri-County
SENER, MD, James P	Wise	SIMPOPOULOS III, MD, Chris	Prince William	SNIDER, MD, John Schurr	Hampton	STEFANIK, MD, Peter J	Residents
SENER, MD, Thos Paul	Out Of State	SIMPSON III, MD, Frank B	Fairfax	SNIR, MD, Arie N	Fairfax	STEFANINI, MD, Mario	Tazewell
SERPHAM JR, MD, Taweesuk	Southside Va	SIMPSON, MD, Geo Winslow	Norfolk	SNODDY, MD, John Wm	Richmond	STEFFEY, MD, Wm Rue	Newport News
SERVIDO, MD, Joseph G	Fauquier	SIMPSON, MD, Melvin Ross	Newport News	SNODDY III, MD, Christopher	Loudoun	STEHLIK, MD, John Mc Teer	Virginia Beach
SESSOMS, MD, Geo Wm	Lynchburg	SIMS, MD, Angela Valencia	Richmond	SNYDER JR, MD, Stanley O	Norfolk	STEIER, MD, Howard C	Norfolk
SETJIRAVIROJ JR, MD, Prasert	Southside Va	SIMS, MD, Arthur I	Arlington	SNYDER, MD, Bernard Melvin	Fairfax	STEIGER, MD, Wm Anthony	Williamsburg
SETTER, MD, John Geo	Portsmouth	SIMS, MD, John Adrian	Alexandria	SNYDER, MD, Bertram C	Arlington	STEIGMAN, MD, Carmen Kay	Residents
SETZLER, MD, Geo Breaker	Out Of State	SIMSARIAN, MD, James Parsons	Fairfax	SNYDER, MD, David Michael	Culpeper	STEIN JR, MD, Jerome	Alexandria
SEXTON, MD, Jo Anne	Lynchburg	SINCLAIR, MD, Terry Louis	Northern Va	SNYDER, MD, James L	Alleghany	STEIN, MD, Charles A	Culpeper
SHACOCCHIS, MD, Thomas J	Newport News	SINGER, MD, Jerry R	Residents	SNYDER, MD, Roger Alan	Fairfax	STEIN, MD, Donald Underwood	Fairfax
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SHAFFER, MD, John S	Southwest Va	SINGH, MD, Harinder Paul	Richmond	SOIFER, MD, Edgar Henry	Fairfax	STEINBERG, MD, Richard A	Arlington
SHAFFER, MD, Stephen R	Alexandria	SINGH, MD, Kanwal S	Richmond	SOKOL, MD, Richard Andrew	Norfolk	STEINGASZNER, MD, Laszlo C	Prince William
SHAH, MD, Mukesh H	Residents	SINGH, MD, Mrudula	Fairfax	SOLAN, MD, Stuart Mile	Richmond	STENICKA, MD, Francis John	Portsmouth
SHAH, MD, Narendra Champaklal	Roanoke	SINGH, MD, Ram	Wise	SOLANO, MD, Simon	Richmond	STENSLAND, MD, Mark S	Richmond
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SHANHOLTZ, MD, Mack Irvin	Richmond	SISK, MD, Michael Anthony	Roanoke	SOLINAP, MD, Perla Juaneza	Norfolk	STEPHENS, MD, Wesley G	Roanoke
SHANK, MD, David Lee	Rockingham	SISMANIS, MD, Aristides	Richmond	SOLOMON, MD, Jonathan G	Newport News	STEPHENSON, MD, John Aldrich	Lynchburg
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SHAPIRO, MD, Andrew Dishart	Roanoke	SITES, MD, James G	Fairfax	SOLTANY, MD, Ray A	Fairfax	STERN, MD, Eric	Fairfax
SHAPIRO, MD, Carol Sadie	Prince William	SJOLUND JR, MD, Geo Clarence	Virginia Beach	SOMERS, MD, Lewis Frank	Lynchburg	STERNLIGHT, MD, Ludwig	Virginia Beach
SHAPIRO, MD, Jerome Jos	Fredericksburg	SKAGGS, MD, Jerome D	Southside Va	SOMMERS, MD, Geo Walker	Out Of State	STEVENS JR, MD, Ward Wm	Roanoke
SHAPIRO, MD, Sam Leon	Norfolk	SKANSI, MD, Tom Andrew	Virginia Beach	SONG, MD, Soon Bock Kim	Lynchburg	STEVENS, MD, Michael Peter	Fredericksburg
SHARMA, MD, Ashok K	Arlington	SKANSI, MD, Viviana	Norfolk	SONNENBERG, MD, Stephen M	Alexandria	STEVENS, MD, Patricia E P	Fredericksburg
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SHEELY, MD, Wm Edward	Alexandria	SLAYTON, MD, Michael Edward	Southwest Va	SOUTHWORTH, MD, Lawrence E	Fredericksburg	STEWART, MD, Lauraine	Richmond
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SHELLEY, MD, Ronald N	Wise	SLOOP JR, MD, Frank B	Albemarle	SOWERS, MD, Richard P	Richmond	STIBAL, MD, Darlene M	Residents
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SHERMAN, MD, Claude Porter	Patrick-Henry	SMITH, MD, Alfred L	Richmond	SPENCER, MD, Donald Lynn	Out Of State	STOKES, MD, Parker Rea	Hampton
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SHERROD, MD, John Philip	Hanover	SMITH, MD, Bobby L	Norfolk	SPENCER, MD, Harrison C	Southwest Va	STOKES, MD, Thos Lane	Norfolk
SHERRY, MD, John Barry	Augusta	SMITH, MD, Chas Glenn	Arlington	SPENCER, MD, Henry S	Richmond	STOLL, MD, Edward J	Lynchburg
SHESHADRI, MD, Bhagvan	Wise	SMITH, MD, Claude Armistead	Norfolk	SPERBER, MD, Edward Ephraim	Norfolk	STONE III, MD, James B	Southwest Va
SHEVLIN, MD, Wm Anthony	Prince William	SMITH, MD, Crawford C	Richmond	SPERLING, MD, Michael H	Norfolk	STONE III, MD, Wm Leete	Arlington
SHEBARO, MD, Uthman Abd-Salam	Fairfax	SMITH, MD, Dallas Edwards	Newport News	SPERRY, MD, Thos Howard	Virginia Beach	STONE JR, MD, Harry Benj	Roanoke
SHIEH, MD, Frank F	Southside Va	SMITH, MD, David M	Lynchburg	SPETZLER, MD, Bertram	Roanoke	STONE JR, MD, C. A.	Southwest Va
SHIELD JR, MD, James Asa	Richmond	SMITH, MD, J Earle	Richmond	SPIEGEL III, MD, Albert	Fairfax	STONE, MD, Gail R	Southwest Va
SHIELDS JR, MD, Randolph T	Augusta	SMITH, MD, James Alexander	Richmond	SPIERS, MD, Dennis Michael	Portsmouth	STONE, MD, James W	Albemarle
SHIELDS, MD, William J	Newport News	SMITH, MD, James H	Southwest Va	SPILLMAN, MD, James Blair	Southwest Va	STONE, MD, Kearthott Mc Caull	Mid-Tidewater
SHIFLETT, MD, Douglas W	Danville	SMITH, MD, John Randolph	Patrick-Henry	SPINR, MD, Camilla B	Williamsburg	STONE, MD, Wm M	Roanoke
SHIH, MD, Teh-Chang	Alexandria	SMITH, MD, Jos John	Newport News	SPIVEY JR, MD, John Carl	Fredericksburg	STONE, MD, Wm M	Alexandria
SHIM, MD, Chi Yun	Richmond	SMITH, MD, Jos Paul	Norfolk	SPLAN, MD, Thomas Paul	Newport News	STONEBURNER, MD, John M	Danville
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SHIM, MD, Young S	Southside Va	SMITH, MD, Larry F	Lynchburg	SPONAU, MD, Harlan Dale	Portsmouth	STORY, MD, Wm Henry	Rockingham
SHIN, MD, Wan	Fairfax	SMITH, MD, Leroy	Richmond	SPONZO, MD, Robt Wm	Out Of State	STOUT, MD, Robt E	Hampton
SHINER, MD, Philip Thompson	Roanoke	SMITH, MD, Lindley Theodore	Richmond	SPORN, MD, i Norman	Richmond	STOWELL, MD, Jeremy A	Fairfax
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SHOHAM, MD, Myron Alan	Fairfax	SMITH, MD, Mathew Norris	Fairfax	SPRENKLE, MD, Wilson B	Residents	STRAIN, MD, Brian M	Residents
SHORTBRIDGE, MD, Chas M	Roanoke	SMITH, MD, Maurice J Vernon	Richmond	SPRINGALL, MD, Walton H	Out Of State	STRATFORD, MD, Thos Peirson	Richmond
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SHOWALTER, MD, Henry Bernard	Richmond	SMITH, MD, Michael E	Rockingham	SPROUL, MD, A Erskine	Augusta	STRAWINSKY, MD, Elz R Caro	Arlington
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SHOWALTER, MD, Samuel G	Rockingham	SMITH, MD, Nelson Montgomery	Southside Va	SREENIVASAN, MD, Nirmala	Wise	STREICKER, MD, William F	Richmond
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SHUMAN, MD, Lawrence Henry	Alexandria	SMITH, MD, Richard A	Residents	STAHL, MD, Neil Ira	Fairfax	STRINGER, MD, Kenneth Robert	Richmond
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SIBLEY, MD, Wm Langley	Roanoke	SMITH, MD, Samuel	Residents				



**T**

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TALBOTT, MD, William G  
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THOMAS JR, MD, Harry  
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THOMAS, MD, Bruce Richard  
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THRASHER, MD, Patrick D.  
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TISNADO JR, MD, Jaime  
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TOMPKINS, MD, Grover Robt  
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TOMPKINS, MD, William F.  
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TONNESEN, MD, Glenn L  
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TORRE, MD, Anthony Vincent

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Rockingham  
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Richmond  
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Norfolk  
Norfolk  
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Southwest Va  
Richmond  
Roanoke  
Portsmouth  
Southwest Va  
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Augusta  
Northern Neck  
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TORRES-LISBOA, MD, Patricio  
TORRISI, MD, John Russell  
TOSI, MD, Richard Englund  
TOSTER, MD, Michael David  
TOWNE, MD, Chas Henry  
TOWNSEND, MD, Henry Le Roy  
TOXOPEUS, MD, Margaret E  
TRABERT, MD, Richard Eric  
TRALKA, MD, Geo Anthony  
TRAN, MD, De Dinh  
TRAN, MD, Trong Cuong  
TRANT III, MD, John Hill  
TRAVERS, MD, Richard D  
TRAVIS, MD, Thos Roper  
TRAYNHAM III, MD, John E  
TREICHLER, MD, Howard P  
TREMOLS, MD, Guillermo A  
TRICE, MD, Clarry Clyde  
TRICE, MD, Ernest Randolph  
TRICE, MD, Jerry Ashby  
TRICE, MD, Robt Palmer  
TRIESHMANN, MD, Helmut W  
TRIMBER, MD, Connell James  
TRIMMER, MD, Karen Rae  
TRINIDAD, MD, Juan A  
TRIVETTE, MD, George A  
TROIANO, MD, Raymond G  
TROLAND, MD, Chas Edward  
TROSTLE, MD, Thomas F  
TROUP, MD, James B  
TROXEL, MD, Geo E  
TROXEL, MD, James Roy  
TRUE, MD, De Witt Sidney  
TRUEDELL, MD, Frank B  
TSAPOS, MD, Michael John  
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TSOU, MD, Anthony Y  
TSUI, MD, Edward S  
TUAZON, MD, Oscar C  
TUCK, MD, Kenneth Douglas  
TUCKER, MD, Henry Jos  
TUCKER, MD, Stanley Cole  
TUCKER, MD, Weir M  
TUCKER, MD, Wm T  
TUGGLE, MD, M. Stuart W  
TULLOCH JR, MD, Earl F  
TULOU, MD, Nicolas Paul  
TUNNER, MD, Wm Sams  
TURALBA, MD, Cornelius  
TURKEKUL, MD, Fuat  
TURNER III, MD, John Mills  
TURNER III, MD, U.G.  
TURNER JR, MD, James Witche  
TURNER JR, MD, Lewis John  
TURNER, MD, Arthur Alvin  
TURNER, MD, Frank Graber  
TURNER, MD, Franklin C  
TURNER, MD, Jack Cocke  
TURNER, MD, Mary Ann  
TUSING, MD, Thos Wm  
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TWYMAN, MD, James Baker  
TYLER, MD, David  
TYLER, MD, Gilman Rackley  
TYLER, MD, John Hutchinson  
TYNES II, MD, William Vernon

**U**  
UBELHART, MD, Charles Robert  
UENO, MD, Winston Mizuo  
ULLMAN, MD, James Irwin  
ULMER, MD, Jack L  
UMALI, MD, Filemon De Jesus  
UMSTOTT, MD, Charles Edward  
UNDERWOOD JR, MD, Paul B.  
UPDIKE JR., MD, Glenn B  
UPTON, MD, David Leslie  
URENA, MD, D.A.  
URUETA, MD, Enrique E  
UY, MD, Flaviano  
UY, MD, Gregorio C  
UZER, MD, Yuksef

V  
VADEN, MD, Edwin Booth  
VADNEY, MD, Richard Claude  
VAID, MD, Arun K.  
VALDIVIESO, MD, Jorge R  
VALENTI, MD, Branko Sergio  
VALENTINE, MD, Lawrence E  
VALK, MD, Thomas Heyward  
VALONE JR, MD, James Austin  
VAN DEN BRANDEN, MD, F M  
VAN DER SOMMEN, MD, Lynda L  
VAN DER WOUDE JR, MD, H  
VAN DRIEM, MD, Geo Henri  
VAN GERTRUUYDEN, MD, H.H.  
VAN HORN, MD, Chas Newton  
VAN NAME JR., MD, Arthur L  
VAN OSTEN, MD, George K  
VAN REKEN, MD, David E  
VAN WOLKENTEN JR, MD, R  
VANCE JR., MD, John Clair  
VANCE, MD, Douglas Doriot

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## Triathlon fever rises in Virginia

Now that the marathon has become a crowd event, fitness buffs are turning to the non-Olympic discipline known as the triathlon.

The triathlon combines running, biking and swimming; each of these segments can be specified in any length the local arrangements committee chooses.

The Ironman Triathlon in Hawaii, for instance, takes the triathlete through a swim of 2½ miles, immediately followed by a bicycle race of 100 miles, and then, with no rest, a regulation marathon of 26 miles.

The Sandman Triathlon in Virginia Beach, on the other hand, lets you off with a one-mile swim, 16-mile bike ride, and 6.2-mile run.

The exigencies of locale produce other variations on the triathlon theme. In both the Ironman and the Sandman, the competitors swim in the ocean, but in the Richmond Triathlon the swimmers lap a pool, and in the Virginia Triathlon at Altavista and the Reston Triathlon in Northern Virginia, the triathletes swim in lakes. Similarly, a triathlon's running segment may be over relatively flat urban streets, as in the Richmond Triathlon, or up hill and down dale, as at Altavista.

Whatever the degree of difficulty, if it's mortification of the flesh you're after, the triathlon fills the bill. In last year's Sandman, hypothermia plagued the ocean swimmers so severely that some had to be pulled out of the water and several were hospitalized. The bike race brought one broken collar bone and a rash of cuts and scrapes. Here and there competitors collapsed from fatigue.

Everyone who crossed the finish line was a case study in the over-use syndrome.

"And these are extremely well-motivated, well-trained people," emphasizes Dr. Clarke Russ of Virginia Beach, the Sandman Triathlon's medical director. "They train at least two to four hours a day, some of them a lot more. You have to make a major commitment."

All of the injured recovered quickly, he points out, partly because an abundance of emergency personnel and equipment lined both land and water routes and partly because the triathlete's "cross-training" in three sports exercises all three of the body's major muscle groups, thus yielding a comprehensive physical fitness.

Of the Sandman's competitors last year, none was fitter than Dr. Rudolf F. Schuster of Norfolk. Rudi Schuster is a 57-year-old family physician who has been competing in triathlons for four years. He covered the Sandman course in 2 hrs 9 mins. The winner was a 30-year-old Marine from Quantico who crossed the finish line in 1 hr 54 mins. In his age group, Dr. Schuster won handily, as he does with such regularity that he's said to be the fastest 50-year-old on the East Coast's triathlon circuit.

He leaves many younger triathletes in his wake as well—for instance, Dr. Michael J. Hechtkopf, 37-year-old Virginia Beach dentist who founded the Sandman meet and is no slouch as a triathlete himself. When the two competed recently in the Oxford, Maryland,

triathlon (2-mile swim, 50-mile bike race, 20-mile run), Rudi Schuster beat Mike Hechtkopf by seven minutes.

"Twenty years older than I am and he beat me, can you believe that?" says Dr. Hechtkopf. He and the physician talk of doing the Ironman together some day. "He'll probably beat me," forecasts the dentist, who carries a daunting mental image of competitor Schuster. "Normally, a couple of friends passing, you'll joke a little with each other, but when Rudi passes you, he is *serious*, man, he doesn't say a word."

Dr. Schuster trains seriously, too. He began running in 1970 and runs "all around Norfolk," as he puts it, but likes especially to jog over the dunes of Seashore State Park, west of Norfolk on Cape Henry, where the Chesapeake Bay meets the Atlantic Ocean.

His first footrace was the Virginia Beach one-mile run. "I was sore for a week," he remembers. Gradually he worked up to marathon competition, running the Boston Marathon twice, but what he calls a "sciata/disc problem" developed, and an ailing Achilles tendon had to have surgery, so he turned to triathlon competition. "Lots of runners with injuries who no longer can run long distance can handle the triathlon," he explains.

Biking he began during his formative years in Germany, where he was born and where he earned his MD at the medical school of Ludwig Maximilians University in Munich. An excelling bicyclist, he competes



# NEWS BUREAU

in bike races all over the country and has twice placed second in the Grandmasters National Bicycling Championships. This month he will go to Colorado for the 1984 Championships to try once again for a first. A week later he'll be charging into the surf off Virginia Beach as the gun goes off for this year's Sandman Triathlon.

In preparation for these two meets triathlete Schuster is training three to four hours daily for six days of the week; on the seventh day he rests. How does he find time for all the training and travelling? "You just make the time," he says. As a solo practitioner, he can bend his office schedule to accommodate, and since he is not married, his after-office hours are his to command. A colleague covers for him when he's out of town for a race.

The unremitting physical regimen has paid off in a body that is the envy of his peers. "Lean, hard, in incredibly good shape," is the way

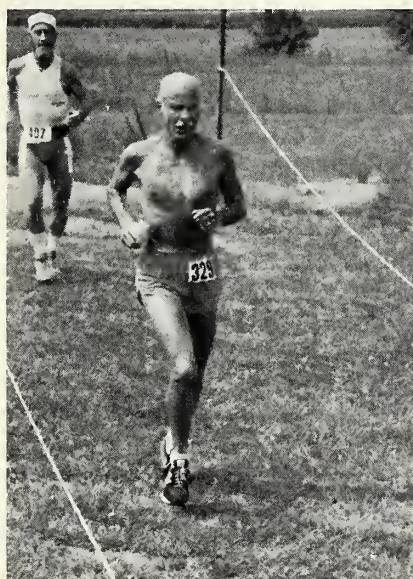
one doctor describes him. "He has the physique of an 18-year-old," avers another. Toiling through the 1983 Sandman without mishap, Dr. Schuster afterwards complained only of "getting beat up right and left" in the ocean's heavy swells and experiencing an irritating side-ache during the run.

Such safe passage is a tribute to his endurance, for the triathlon is a punishing grind with physical hazards unique to its three-part formula and injuries that bring a new set of biomechanics into the doctor's office. To explore these medical aspects, Dr. Russ, an orthopedist and sports medicine specialist, has initiated the first national conference on the triathlete, "Runners, Bikers, Swimmers", to be held on September 22 in Virginia Beach. Serving

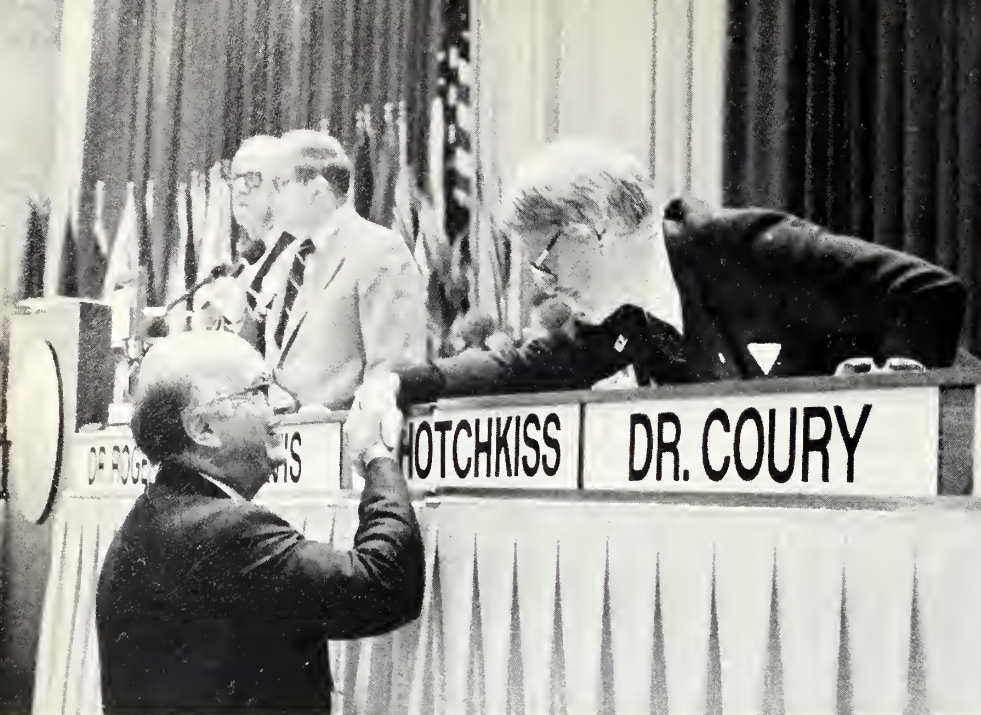
with him on the course committee are Dr. Paul N. Krop, also an orthopedist; Dr. Richard A. Mladick, plastic surgeon, marathon runner and triathlete; and Dr. J. Timothy Devlin, family physician and bike racer. All of them practice in Virgin-



**Dr. Rudi Schuster competing in the three phases of the Oxford, Maryland, triathlon.**







Joe Fletcher for the AMA

**Winner:** Dr. William S. Hotchkiss, Chesapeake, leans forward to accept the congratulations of Dr. Daniel T. Cloud of Phoenix, Arizona, after the Virginia physician's reelection to the American Medical Association's board of trustees during the AMA's annual meeting in mid-June in Chicago. President of The Medical Society of Virginia in 1971-1972, Dr. Hotchkiss has been an AMA trustee since 1980, has served as the Association's secretary-treasurer, and is an AMA commissioner to the Joint Commission on the Accreditation of Hospitals. He is a thoracic surgeon. Dr. Cloud is a past president of the AMA.

ia Beach. Dr. Mladick is president of the Virginia Beach Medical Society; he shares with Dr. Russ the medical directorship of the Sandman Triathlon and will be taking that duty this year.

The conference originally was titled "Triathlete: Training, Trauma, Treatment," but then Dr. Russ discovered a lot of physicians had not yet heard about this new fitness fever. This is not surprising, since the triathlon first appeared only nine years ago in San Diego and from there spread westward to Hawaii, where the Ironman was launched in 1977. Two years later, the Ironman in all its gruelling glory was shown on national television, and triathlons began popping out all over the country.

Today there are two national magazines devoted exclusively to triathletics and a national sanction-

ing organization, Triathlon Federation USA, or TriFed, which, a spokesman told this reporter, has given official assent this year to upwards of 600 triathlons over the country.

In Virginia, the first triathlon appeared in 1981, in Richmond; this year there were five, with a total enrollment of 2,150 runners-bikers-swimmers. The Virginia Triathlon at Altavista accommodates 800 of that total in an unusual format combining individual competitors and three-person teams. In Virginia Beach, 450 will jostle through the Sandman; 400 sweated through the Richmond meet; and the Ocean View Triathlon in Norfolk, new this year, drew 300 contestants.

Another 1984 newcomer, the Reston Triathlon, planned for 125 entrants, expanded to accept 200, and still turned 100 away; that's the

standard triathlon enrollment pattern. "There just aren't enough triathlons in Virginia to go around," laments one official.

To be sure, triathlons probably will never attract the contestants in numbers rivalling the marathons. For one thing, three skills must be mastered, rather than one. Second, for many, swimming facilities are hard to come by. Third, the logistics of triathlon competition exact additional demands—getting the gear together, mastering the mechanics of a bike, engineering swift changes of gear and clothing.

Even so, the triathlon enthusiasm is big and getting bigger, and triathletes predict that one year soon they will be given the Olympic seal of approval.

—ANN GRAY

## Commissioner explains tax liability

After several physicians called Medical Society of Virginia headquarters with questions about Virginia's sales tax, State Tax Commissioner W. H. Forst was asked for information to explain the Virginia retail sales and use tax as it applies to the practice of medicine.

His response began by setting forth exemptions to the sales and use tax. Section 58-441.6(s) exempts "medicine, drugs, hypodermic syringes, artificial eyes, contact lenses, eyeglasses and hearing aids dispensed by or sold on prescription or work order of licensed physicians, dentists, optometrists, ophthalmologists, opticians, audiologists, hearing aid dealers and fitters, and controlled drugs purchased by a physician for use in his professional practice."

Section 58-441.6(s1) exempts these from sales and use tax: "Wheelchairs and parts therefor, braces, crutches, prosthetic devices, orthopedic appliances, cath-



eters, urinary accessories, insulin and insulin syringes, when purchased by or on behalf of an individual for use by such individual."

The Commissioner was careful to point out that according to the Virginia Department of Taxation's interpretation, these exemptions apply only to purchases by patients and do not apply to purchases by physicians. The only items a physician may purchase tax-free for use in his practice are the "controlled" drugs of Schedules I-VI, including injectible penicillin, oral polio vaccine, vitamin B<sub>12</sub>, injectible gold for arthritis, and phergran. The department does not consider devices,

such as diaphragms and tuberculin time testers, to be within the scope of the statutory exemption for controlled drugs, Commissioner Forst added.

He pointed out that a physician who holds a special certificate from the Board of Pharmacy and who buys and sells drugs, in effect acting as a pharmacist, may purchase items for resale exclusive of the sales tax when there is no drug store in the community; the use tax should be remitted by such a physician, however, if any such items (except controlled drugs) are withdrawn from his resale inventory to be used in his practice.

A. Clement, director of the Health Department's Division of Health Planning.

"We're very upset," Dr. McDowell told VIRGINIA MEDICAL's reporter. "We were so hopeful, when all three of the reviewing boards endorsed the proposal."

Would the orthopedists go on to the second, formal appeal? They hadn't decided, Dr. McDowell, said. They'd have to hire legal counsel at a fee that could reach \$10,000, and it looked like a risky investment.

Meantime, after protracted negotiations Dr. Nelson was granted a license for the urologists' free-standing ASC, and it opened for business on May 16. How's it going? "Doing nicely," in Dr. Nelson's words.

Both Dr. Nelson's group and that of Dr. McDowell practice in Planning District 15 of the Central Virginia HSA area. There are five short-stay hospitals in PD 15. All five have ambulatory surgery programs.

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## More (mis)adventures in CON country

Dr. C. M. Kinloch Nelson of Richmond, whose bout with the certificate of need system was chronicled in VIRGINIA MEDICAL's November 1983 issue, has returned to the fray in quest of another free-standing ambulatory surgery center.

Last year his petition sought an ASC for himself and his six associates in urology. After a bureaucratic adventure rivalling the Perils of Pauline, CON artist Nelson won approval.

This time the facility Dr. Nelson is proposing would be used by five specialties: general surgery, gynecology, orthopedics, urology and ear-nose-throat.

The new application cleared its first hurdle when it was approved by the board of directors of the Central Virginia Health Systems Agency, who at the same meeting also endorsed a petition for a free-standing eye surgery center brought by three Richmond, ophthalmologists, Dr. Anthony D. Sakowski, Jr., Dr. Walter E. Bundy III, and Dr. J. Paul Bullock, Jr. Earlier this year the board had given its assent to an application for an ASC

brought by six Richmond surgeons who are associated in orthopedic practice: Dr. Charles L. McDowell, their president, Dr. John D. Bowman, Dr. Edward P. Carrigan, Dr. Daniel A. Dethmers, Dr. Joseph R. Macys and Dr. Richard L. Worland.

Other applications are said to be in preparation, in what one HSA staff member termed "an explosion" of requests for these physician-owned facilities.

The petitioners are not likely to have any easy time getting past the upper echelons of the certificate of need system. In a replay of Dr. Nelson's experience last year, the orthopedists' application was denied by State Health Commissioner James B. Kenley. Then the scenario changed: when Dr. Nelson appealed the Commissioner's denial, the appeals officer recommended reversal and Dr. Kenley complied, but when the orthopedists appealed, the Commissioner's denial was upheld. The appeals officer at the urologist's hearing was Dr. Bedford E. Berrey, Assistant State Health Commissioner. Dr. McDowell's appeal was heard by Samuel

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## Robb names MDs

Two Medical Society of Virginia members have been appointed to state posts by Gov. Charles S. Robb.

Dr. Gerald C. Burnett, South Hill, was named to the Virginia State Board of Medicine. Chairman elect of VaMPAC, Dr. Burnett is The Medical Society of Virginia's vice councilor for the 5th district and chairman of the Pharmacy Committee. He is an allergist.

Dr. Henry S. Campell, Martinsville, was appointed to the State Board of Social Services, the body that sets policy for social services throughout Virginia. An ophthalmologist, Dr. Campell has long served as a delegate to MSV meetings from the Patrick Henry Medical Society and is a member of VIRGINIA MEDICAL's Editorial Board.

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## From the President: The American Way

**A**s I returned from this year's Fairfax City's 4th of July parade, I started thinking: What could be more American—an event and public turnout that encompasses young people and old, families, grandparents, native-born and ethnic groups of all types. This is the kind of experience and public participation that epitomizes America and makes me proud of our heritage and proud to be an American.

On returning home my thoughts wandered to medicine and politics and I became slightly depressed. I thought of how important physician activism is in both these areas and how few doctors become involved in the political process. Many physicians feel that politicians are two-faced and frequently compromise their positions in order to obtain part of the goal rather than the entire goal. This may be because we as physicians are taught from the beginning that arriving at the correct diagnosis and helping the patient is our goal *without compromise*. It also may be a reason for not participating.

Whatever the reason, it's distressing that many doctors and their families are not even registered to vote. The percentage in that category reaches 40% in some areas of the country. If doctors, who comprise one of the highest educated groups in the country, are apathetic about exercising their rights as citizens, they shouldn't complain about the results of local and national elections or what course this country takes.

Some physicians I know do hold political office, do work and do contribute to the parties of their choice. And all physicians should be grateful for that. At the same time, I want to encourage those of you who do little or

nothing to become more actively involved. I have heard all the stories: you don't have the time, you forgot, or none of the candidates are any good. However, these are not satisfactory responses for not exercising your inalienable right to vote. The AMA has launched a project that I firmly believe in. It's called MEDVOTE and it is working to encourage physicians and their families to register and vote.

Registering to vote and participating actively in the political process is an important responsibility of all physicians. As individuals we have much to contribute to the quality of this process; as a group our influence can be even stronger.

The AMA also understands the importance of group strength and I urge all Virginia physicians to seriously consider joining the AMA. Only about 50% of physicians currently belong to the AMA. But where else can you get as much for the money? The AMA has a strong political lobby on Capitol Hill which works to promote and protect the quality of medicine in this country. It also is the largest medical scientific publisher in the world.

The leadership of the AMA and The Medical Society of Virginia is deeply concerned about people and the quality of the health care they receive. Organized medicine is working to assure that we will be able to give quality care in the future. If for no other reason, you should join the AMA if you are concerned about your patients and all of the patients of this country, so we can work together to insure quality care for all. Remember, your involvement *can* make a difference.

C. BARRIE COOK, MD, PRESIDENT  
The Medical Society of Virginia

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# ABSTRACTS

*These abstracts were selected from those presented at the annual meeting of the Virginia Society of Hematology and Oncology on June 22 and 23, 1984, at Virginia Beach.*

**Use of Intravenous Gamma Globulin in Acute Idiopathic Thrombocytopenic Purpura.** Michael G. Levien, MD, Hernan Sabio, MD, Thomas H. Howard, MD, Debra D. Conrad, MD, Marwan Hanna, MD, and Louis A. Vega, MD, *Charlottesville.*

This is a report of two pediatric patients with aggressive, potentially life-threatening bleeding secondary to idiopathic thrombocytopenic purpura (ITP) treated with high dose IV gamma globulin with subsequent rapid rise of platelet count to protective levels. Patient #1 presented with severe epistaxis. He also had multiple hemorrhagic varicella lesions. Prednisone was not used due to active varicella. Patient #2 also had epistaxis and significant hematuria. He initially was started on Prednisone. Both patients were given IV gamma G 600 mg/kg IV q day  $\times$  3 days with subsequent rapid rise of platelets. Both patients had positive indirect

platelet antibodies. Platelet counts were as follows:

Platelet counts	Day #1	Day #2	Day #3	Day #4	Day #7
Patient #1	3000	32,000	113,000	123,000	63,000
Patient #2	4000	101,000			663,000

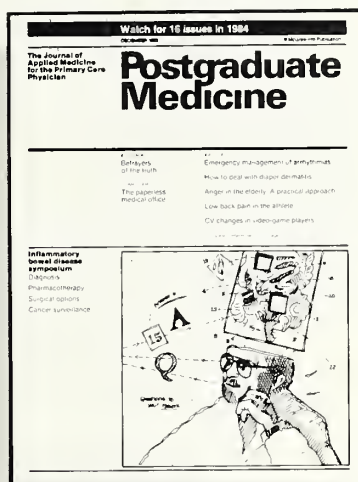
Patient #1 developed rebound thrombocytopenia with nadirs of 11,000 at 2 weeks requiring a course of steroids with subsequent resolution of thrombocytopenia. Patient #2's platelets remained normal after IV gamma G was stopped on day #3. There is a definite role for using IV gamma G in acute ITP with significant hemorrhagic manifestations.

**Pulmonary Complications Following 1,3-Bis(2-Chloroethyl)-1-Nitrosourea Therapy (BCNU).** G. M. Mire, MD, and G. G. deBlois, MD, *Richmond.*

Pulmonary fibrosis has recently been described as a complication of BCNU therapy. We report a

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**In Vitro Oxygen Uptake ( $\dot{V}O_2$ ) and Mitochondrial Density in Hamster Striated Muscles.** Sharon Sullivan Camden (M-86) and Roland N. Pittman, PhD.

The research presented here represents measurements of oxygen consumption and numerous anatomical parameters which may influence oxygen transport to tissue in muscle fibers of different histochemical fiber type. In vitro oxygen consumption ( $\dot{V}O_2$ ) was measured in the retractor (RET) muscle, a mixed skeletal muscle (70% fast-twitch-glycolytic, FG; 16%, fast-twitch-oxidative-glycolytic, FOG; 14% slow-twitch-oxidative, SO) as well as two other muscles in the hamster, the soleus (SOL) and sartorius (SAR) muscle. The SOL and SAR muscles were chosen because they are essentially composed of either oxidative fiber types (57% FOG, 43% SO) or glycolytic fiber types (98% FG, 2% FOG), respectively, and data from these two muscles were used to estimate single fiber oxygen consumption in the retractor muscle. In addition, electron micrographs were taken in each of these three muscles and anatomical parameters such as fiber area, surface area/volume ratio, mitochondrial

density, number of capillaries around a muscle fiber and capillary per fiber membrane contact length were measured. Results indicated that:  $\dot{V}O_2$  in the SOL ( $1.0 \pm 0.048$  (SE)  $\text{ml O}_2 \cdot \text{min}^{-1} \cdot 100\text{g}^{-1}$ ) was significantly different ( $p < 0.01$ ) from  $\dot{V}O_2$  in the RET ( $0.89 \pm 0.038$ ) or SAR ( $0.80 \pm 0.034$ ) muscles. Oxidative muscle fibers, in general, were smaller in cross-sectional area, had a larger number of surrounding capillaries and had greater surface area/volume ratios when compared to glycolytic fibers. Mitochondrial density (measured using stereologic methods) was greatest in the SOL ( $0.18 \pm 0.009$ ), least in the SAR ( $0.09 \pm 0.008$ ) and of intermediate value in the RET ( $0.12 \pm 0.008$ ). Hopefully such information can be used to further our knowledge in the area of oxygen transport to tissue.

**Morphologic and Physiologic Studies of Thymic Epithelial Cells.** Jeffery K. Taubenberger (M-86).

The role of the thymus in T-cell development has been well documented. Histologic examination of the thymus demonstrates a complex architecture, with a stroma consisting of at least three cell types: epithelial cells, macrophages, and fibroblasts. To study the role that the thymic epithelial (TE) cells play in T-cell maturation, cultures of these cells

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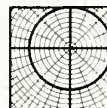


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were established. Thymuses from newborn CBA mice were minced and cultured in D-valine containing media to suppress fibroblast overgrowth. Cells were later grown on cytodex beads. Stable cultures of a dimorphic cell population were established. Morphologic studies with light and electron microscopy revealed these cells to be epithelial cells and macrophages. The in vitro chemo-attractant activity of these cells was evaluated by blind well migration assays, performed with supernatant from these cells and bone marrow (BM) cells. The migration index (% of BM cells migrating) was determined and shown to be significantly higher than control cells. The cells collected with the migration assay were labeled with FITC and injected into irradiated mice. A cell suspension of the host thymus at 4 and at 16 hours when analysed by flow cytometry revealed a higher level of fluorescence than controls, demonstrating an enrichment for prothymocytes from the BM pool. To test the in vivo formation of TE cell cultures, streaker mice (congenitally athymic) were injected subcutaneously with TE cell coated beads. At two weeks and at two months, histologic examination and cytotoxic T-lymphocyte (CTL) assays were performed. Sections of the injection site showed outgrowth of TE cells from

the beads, capillary ingrowth and lymphocytic infiltrates. The CTL assay showed negligible chromium release (target cell death) of control mice (no injection; beads alone). However, mice injected with the TE cells at two months showed cytotoxic activity over 50% of normal controls. Thus, the ability of these cells to enrich for prothymocytes as well as enhance T-cell development in vivo has been demonstrated.

#### **A Cellular Pharmacokinetic Study of Methotrexate and Its Polyglutamate Metabolites in CF-1 Mice.** Matthew Borst (M-84).

A growing body of in vitro data from a variety of cell types suggests that the metabolism of methotrexate (MTX) to polyglutamyl derivatives (MTX-PGs) may be an important determinant of cytotoxicity during MTX chemotherapy. In this study, the rates of synthesis and retention of the polyglutamate metabolism of MTX were studied in the Ehrlich ascites tumor cell line and selected tissues (plasma and GI mucosa) of CF1 mice in vivo to determine whether there are differences in this metabolic conversion between tumor and susceptible host tissues that may be a basis for drug selectivity. When tumor bearing mice received sub-

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cutaneous injections of MTX (2 mg/kg) the intracellular peak concentration of drug was reached within two hours in each tissue studied. HPLC analysis revealed intracellular drug to be comprised of both MTX and MTX-PGs. Between two and five hours after injection, the PG metabolites continued to accumulate in tumor cells and liver as the plasma levels of MTX declined. Cell MTX levels paralleled that of the plasma levels and fell to low levels between four and six hours at a time when intracellular MTX-PGs which had accumulated were sustained during the next 12–16 hours. GI mucosal cell

kinetics revealed that total intracellular drug accumulation rose and fell in parallel with free MTX and exhibited negligible metabolism or accumulation of PG derivatives. These data suggest that PG metabolites of MTX, which are known to have affinity for dihydrofolate reductase (DHFR), accumulate in tumor cells but not in GI mucosal cells, to sustain drug effects for longer in the tumor than in this susceptible host tissue. The rise and fall of the monoglutamate of MTX is the same in both tissues. Polyglutamylation may be an important factor in drug selectivity.

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- A review of the diagnostic features and the clinical and medical implications of anorexia and bulimia;
- A discussion of cognitive behavioral treatment modalities; and
- The presentation of case studies by the speakers and by other symposium participants.

The speakers will be: William L. Webb, Jr., M.D., Vice President and Medical Director, Sheppard and Enoch Pratt; Paul Garfinkel, M.D., Psychiatrist-In-Chief, Toronto General Hospital; and David Roth, Ph.D., Assistant Director Special Problems Unit, Sheppard and Enoch Pratt.

This symposium—one of many educational events sponsored by The Education Center at Sheppard Pratt—is designed for psychiatrists, physicians, psychologists, nurses, social workers and other professionals concerned with the treatment of eating disorders.

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# Recent Advances in Diagnosing and Treating Heart Failure

Andrea Hastillo, MD, P. K. Mohanty, MD, A. Scott Mills, MD,  
and Michael L. Hess, MD, *Richmond, Virginia*

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The spectrum of congestive heart failure has changed, with more cases being reported of non-rheumatic valvular disease, ischemic cardiomyopathy without angina, and dilated congestive cardiomyopathy. Diagnostic and therapeutic techniques have changed, too, but heart failure still carries a two-year mortality rate of 66%.

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**H**EART FAILURE is the inability of the myocardium to deliver an adequate cardiac output to meet the metabolic needs of the body. Congestive heart failure refers to the clinical syndrome characterized by shortness of breath, dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea and peripheral edema due to the left ventricular failure.

The growing importance of the problem of congestive heart failure in the United States can be seen by the increasing incidence of the disease process over the last ten years. In 1970, there were 150,000 discharges from hospitals in the United States with a diagnosis of congestive heart failure, a

77.5 per 100,000 population incidence of the disease. By 1980, there were 401,000 discharges from hospitals in the United States with a diagnosis of congestive heart failure, an incidence of 180 per 100,000 population.<sup>1</sup> The increase in the incidence and the frequency of the disease process leading to heart failure are not well understood but are probably contributed to by the increase in life expectancy, advances in medical and surgical therapy for cardiac diseases that would otherwise have resulted in an early death, and better medical therapy for those patients already suffering from congestive heart failure.

The etiological spectrum of congestive heart failure in the United States has also significantly changed. The decreased incidence of rheumatic heart disease and the presentation of congestive heart failure due to rheumatic valvular disease have become the exception rather than the rule. More common causes of congestive heart failure due to valvular disease are now the entities of calcific aortic stenosis and mitral regurgitation due to myx-

From the Department of Medicine, Division of Cardiology (Drs. Hastillo and Hess), and the Department of Pathology (Dr. Mills), Medical College of Virginia, and the Department of Cardiology, McGuire Veterans Administration Medical Center (Dr. Mohanty). Address correspondence to Dr. Hess at Box 281, MCV Station, Richmond VA 23298.

Submitted 10-12-83.



omatous degeneration of the mitral apparatus. The other two major etiologies include the ischemic cardiomyopathy syndrome and dilated congestive cardiomyopathy. Ischemic cardiomyopathy is characterized by diffuse coronary artery disease with or without previous myocardial infarction and presents as congestive heart failure rather than angina pectoris. Dilated congestive cardiomyopathy falls under the category of idiopathic cardiomyopathy, which is a primary disease of cardiac muscle.

The end result of these three broad categories of disease processes (degenerative valvular disease, ischemic myocardial disease and idiopathic cardiomyopathy) is the same: an inability of the heart to sustain an adequate cardiac output. The final common pathway of a decreasing cardiac output activates several compensatory mechanisms that lead to the presenting signs and symptoms of the patient with congestive heart failure.

### **Pathophysiology**

There are four major factors that determine cardiac output: heart rate; preload (left ventricular end diastolic pressure or left ventricular filling pressure); afterload (the resistance that the heart must overcome to pump blood, i.e., total peripheral resistance); and contractility. With the decrease in cardiac output as a result of impaired left ventricular function, these four factors are brought into play as mechanical attempts to compensate. Low cardiac output in heart failure is associated with reduction in arterial pressure and increased left atrial pressure due to increased left ventricular end diastolic pressure (LVEDP). These changes result in decreased arterial baroreceptor and cardiac sensory receptor activity, which produces a reflex increase in sympatho-adrenal drive and peripheral vasoconstriction. This increased sympathetic activity may be viewed as a compensatory mechanism which tends to augment the depressed contractility and produces the resting tachycardia commonly seen in patients with congestive heart failure. The decreased cardiac output results in a decreased renal blood flow, which activates the renin angiotensin system. With activation of the renin angiotensin system, there is an increased circulating level of angiotensin II and an increased concentration of aldosterone. The mineralocorticoid action of aldosterone acts on the kidney to increase sodium and water reabsorption. This, together with the decreased glomerular filtration rate, results in an increase in sodium and water retention. This mechanism results in the total body increase in sodium and water commonly seen in patients with conges-

tive heart failure. Thus the presentation of the patient with peripheral edema and congested lungs is directly related to an attempted renal compensation for the low cardiac output. This attempted compensation by hypervolemia is the basis of the Frank Starling mechanism, which increases left ventricular end diastolic pressure in an attempt to increase cardiac output. Obviously, this compensatory mechanism is limited by the pulmonary capillary hydrostatic-oncotic gradient of 10 to 20 mmHg. When left ventricular end diastolic pressure exceeds 25 to 35 mmHg, the increase in pressure is directly reflected in the pulmonary capillary system, which in turn results in a transudation of fluid from the pulmonary capillaries to the interstitial space, interstitial edema and, if uncompensated, pulmonary edema.

The signs and symptoms of the patient presenting with congestive heart failure are a direct result of the attempted compensatory mechanisms. Low cardiac output causes reduced systolic blood pressure that one sees in patients with congestive heart failure. The normal to elevated arterial diastolic pressure and narrow pulse pressure are due to an increase in total peripheral resistance that is mediated by increased levels of angiotensin, increased sympathoadrenal drive and vasoconstriction. The low pulse pressure is a direct reflection, therefore, of an increase in total peripheral resistance. The increase in total peripheral resistance, in turn, increases the impedance to left ventricular ejection and further contributes to the lowering of cardiac output. Finally, the increase in the sympathetic nerve activity, which tends to increase cardiac contractility, is a limited compensatory mechanism because of the underlying disease process.

Therefore, the compensatory mechanisms of heart failure are limited and, if unchecked, will further aggravate the congestive heart failure syndrome. The resting tachycardia contributes to an increase in myocardial oxygen consumption and is deleterious, especially in patients with coronary artery disease. The attempted volume compensation limits the patient by the development of pulmonary congestion and peripheral edema. Finally, the activation of sympathetic innervation and renin-angiotensin system directly contribute to the increase in total peripheral resistance. Thus, these attempted compensations can contribute to a circle of positive feedback, which can result in further aggravation of the congestive heart failure syndrome.

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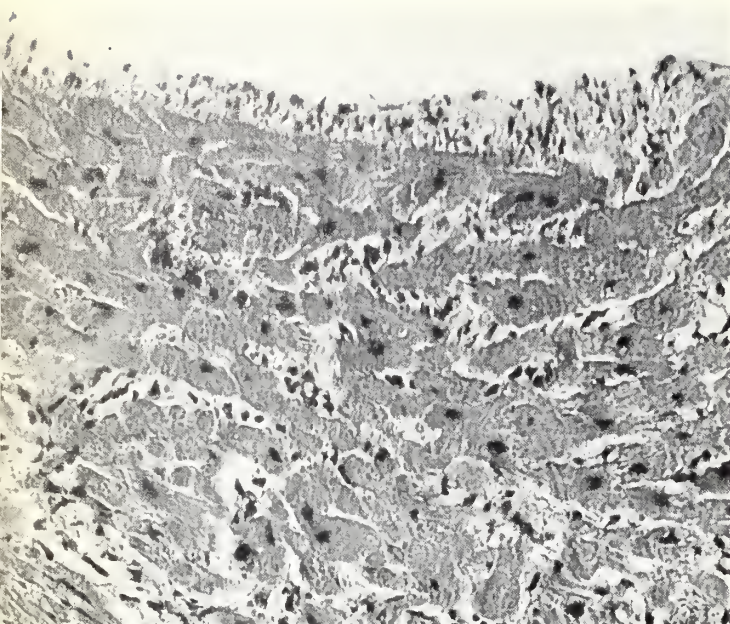


Fig. 1. Endomyocardial biopsy of right ventricle: myocarditis. Endocardium at top. Heavy infiltrate of lymphocytes between myofibers. (H&E  $\times 100$ )

### Diagnosis

The diagnosis of congestive heart failure is still based on an accurate clinical history and physical examination. The patient often presents with a resting tachycardia, a low systolic blood pressure, narrow pulse pressure, rales at both lung bases, an S3 gallop and peripheral edema. The electrocardiogram usually shows a resting sinus tachycardia; if left ventricular hypertrophy or intraventricular conduction defects are present, this will herald a poor prognosis.<sup>1</sup> Chest X-ray will show cardiomegaly with a predominance of the left ventricular silhouette and pulmonary venous hypertension. The echocardiogram has become an accurate office tool for the confirmation of the diagnosis of a dilated congestive cardiomyopathy. The echocardiogram will show dilated cardiac chambers with a significant increase in left ventricular end diastolic and end systolic dimensions and hypodynamic left ventricular wall motion with or without regional wall motion abnormalities. Accurate quantitation of heart failure can be obtained by the gated blood pool study (MUGA), a radionuclide study which can accurately estimate left ventricular ejection fractions. By convention, a left ventricular ejection fraction of less than 40% (normal range of 50%-60%) is taken as indicative of a cardiomyopathic process with compromised left ventricular function. A complete right and left heart catheterization will confirm the presence of elevated intracardiac pres-

ures and will provide an accurate diagnosis to help distinguish between the ischemic cardiomyopathy of severe coronary disease, valvular disease or a primary cardiomyopathic process.

### Right Ventricular Endomyocardial Biopsy

Unfortunately, there is still a large segment of the patient population with the diagnosis of idiopathic dilated congestive cardiomyopathy. This diagnosis connotes normal coronary artery anatomy, normal cardiac valve anatomy and primary disease of cardiac muscle manifested by a poorly contractile ventricle and low ejection fraction. In an effort to further evaluate these patients and better understand some of the disease processes that affect cardiac muscle, the procedure of right ventricular endomyocardial biopsy has been developed. Aside from cardiac allograft rejection, there are four major indications for endomyocardial biopsy. These include: 1) inflammatory myocarditis; 2) adriamycin cardiomyopathy; 3) restrictive cardiomyopathy (amyloid and hemochromatosis); and 4) intractable ventricular arrhythmias in the myopathic patient. The procedure of right ventricular endomyocardial biopsy is extensively utilized by those centers which perform cardiac transplantation. It has also been applied safely to patients with cardiomyopathy. This procedure presents an additional diagnostic tool to the physician and permits the cardiologist to utilize tissue diagnosis in order to better guide the patient's therapy.

Endomyocardial biopsy has resulted in the finding of about a 20% incidence of inflammatory myocarditis in patients presenting with an idiopathic dilated cardiomyopathy.<sup>2</sup> Inflammatory myocarditis is probably viral in etiology and has an immunological mechanism, which contributes to an inflammatory response and progressive destruction of the myocardium (Figure 1). The myocardium in inflammatory myocarditis is extensively involved with polymorphonuclear and monocytic infiltrate, which is felt to be similar to the inflammatory infiltrate affecting the synovium in patients with rheumatoid arthritis. By applying this analogy, there are several centers in the United States that have treated these patients with aggressive immunosuppression with good short-term results.<sup>3</sup> Unfortunately, no control study on the role of immunosuppression in myocarditis has been performed. This remains an exciting field of future clinical investigation.

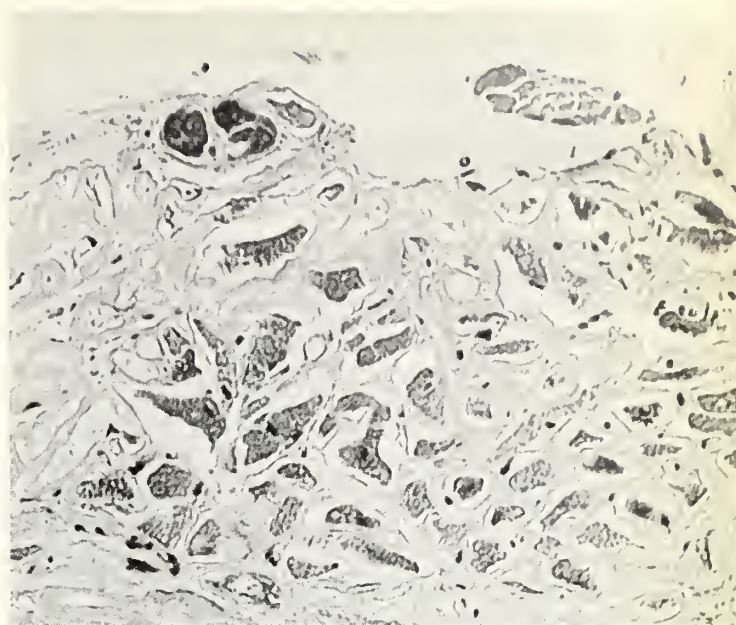
In the diagnosis of restrictive myopathy, endomyocardial biopsy can be of significant benefit to the patient. A common difficult differential diagno-



sis for the cardiologist is the distinction between restrictive cardiomyopathy and constrictive pericarditis. Often a definite diagnosis can only be made by open thoracotomy and visual inspection of the myocardium and pericardium. However, with the advent of right ventricular endomyocardial biopsy, cardiomyopathic process can be identified and a diagnosis of restrictive cardiomyopathy can be established. This can save the patient the risk of open thoracotomy. Figure 2 presents the right ventricular endomyocardial biopsy from a patient who was being considered for pericardial stripping for the diagnosis of constrictive pericardial disease. Hemodynamic evaluation suggested but did not conclusively prove a restrictive myopathic process. This biopsy demonstrates diffuse amyloid infiltration of the myocardium, thereby establishing the diagnosis of a restrictive cardiomyopathy due to amyloidosis. With the documented safety and efficacy of endomyocardial biopsy, it is now preferable to biopsy the heart directly rather than indirect assessments of rectal and/or gingival biopsy when considering the diagnosis of amyloidosis as an etiology to a restrictive cardiomyopathic process. Additionally, endomyocardial biopsy is an important adjunct tool for monitoring adriamycin cardiotoxicity in those patients having to undergo chemotherapy. The adriamycin derivatives are directly cardiotoxic, and the clinical evaluation of these patients at times can be difficult because of previous chemotherapy and underlying tumor process. Unfortunately, the diagnosis of adriamycin cardiotoxicity requires the use of electron microscopy, so this indication is limited in its application. Finally, there is a small subpopulation of patients that present either with sudden death or recurrent ventricular tachycardia and syncope with no significant underlying cardiac pathology identified by routine methods, including right and left heart catheterization. Endomyocardial biopsy may reveal an inflammatory process that is contributing to the life-threatening arrhythmias. Therefore, in the last several years, the concept of tissue diagnosis in cardiology is gaining more importance and is becoming a valuable tool, especially in differential diagnosis of caridomyopathies.

### Therapy

Diuretic therapy remains the mainstay of treatment of symptomatic congestive heart failure. With daily therapy, diuretics cause a reduction in intravascular and extracellular fluid volume, which results in a reduction of venous filling pressure and peripheral edema. By decreasing left ventricular end diastolic pressure, the symptoms of orthopnea



**Fig. 2. Endomyocardial biopsy of right ventricle: amyloidosis. Marked interstitial deposition of amorphous amyloid material ringing muscle fibers. (H&E  $\times 100$ )**

and paroxysmal nocturnal dyspnea are relieved. Potent diuretics, such as furosemide, ethacrynic acid and bumetamide, often cause potassium depletion and hypokalemia, a common predisposing factor for arrhythmias. This is especially true in the patient with congestive heart failure who may be otherwise predisposed to ventricular arrhythmias.

The use of digitalis in congestive heart failure is controversial. One report states that digitalis is beneficial only in an outpatient population if the patients have an S3 gallop.<sup>4</sup> In a subsequent study of hospital-based patients, no significant hemodynamic improvement could be demonstrated due to digitalis therapy.<sup>5</sup> What is well appreciated today is that with severe congestive heart failure, the therapeutic to toxic ratio of digitalis becomes very narrow, and the danger of digitalis-induced arrhythmias very real. Therefore, except for the control of supraventricular tachyarrhythmias, the use of digitalis in severe heart failure is probably not useful. Furthermore, there are several other drugs available which can influence the preload and afterload and are effective in the therapy of congestive heart failure.

The organonitrates (Isordil®, Nitropaste®, Transderm®, nitroglycerin) offer significant advantages by acting as preload reducers and increasing the venous capacitance system. This results in a decreased left ventricular filling pressure, a decrease in left ventricular end diastolic pressure, and relief

of the symptoms of orthopnea, paroxysmal nocturnal dyspnea and dyspnea on exertion.<sup>6</sup> The antihypertensive drug hydralazine has been found to be of benefit in the treatment of congestive heart failure.<sup>7</sup> Hydralazine, by acting as a direct arteriolar vasodilator, will decrease total peripheral resistance, reduce impedance to left ventricular ejection and permit an increase in cardiac output. Unfortunately, because of its potent hypotensive effect and tachyphylaxis, the use of hydralazine becomes limited in patients with congestive heart failure, especially in those patients who present with systolic blood pressures in the range of 80-90 mmHG.

A direct pharmacological approach to interrupting the positive feedback loop during the congestive heart failure syndrome has been provided by the angiotensin converting enzyme inhibitor, captopril. Captopril inhibits the conversion of angiotensin I to angiotensin II and as a result produces both an increase in the venous capacitance system and a decrease in total peripheral resistance. This effect reduces left ventricular filling pressure, relieves the patient of congestive symptoms and decreases the resistance to left ventricular ejection, which results in an increase in cardiac output. Captopril was originally developed as a potent antihypertensive agent, and for this reason the starting dose of captopril should be approximately 6.25 mg three times a day. This requires that either the physician or the pharmacist break the square tablets into four quarters and gradually increase the dose, depending on the response. There is currently good data that demonstrates that captopril results in significant long-term improvement in a patient with congestive heart failure.<sup>8</sup>

Currently under development in the United States are several new classes of compounds aimed at increasing myocardial contractility. A prototype drug is amrinone, a potent cardiotonic agent that resembles a phosphodiesterase inhibitor and results in increased intracellular levels of cyclic AMP. This, in turn, increases myocardial contractility. In short-term intravenous infusions, amrinone has been shown to be a significant positive inotropic agent.<sup>9</sup> It is currently undergoing Phase III testing in the United States for long-term use as a positive inotropic agent. In the next several years, there will be the development of several drugs in this class, which should be of significant benefit to the patient with congestive heart failure.

## Conclusion

Congestive heart failure represents an important and growing problem in the United States. There

are an increased number of patients presenting with congestive heart failure of diverse etiologies that require skill in both diagnosis and management. The unfortunate fact facing physicians is that the diagnosis of an idiopathic dilated congestive myopathy with a reduction in ejection fraction still carries a two-year mortality rate of approximately 66%.<sup>1</sup> The use of vasodilator therapy has been shown to decrease the number of hospitalizations required by these patients but has not significantly improved the two-year survival rate. Thus the challenge to the medical community is to find the combination of medical therapies that will both increase the patient's functional capability and prolong the life expectancy in spite of the presence of a severely compromised myocardium. To date, the only other alternative that exists is cardiac transplantation, which is still limited in its application and fraught with a great deal of difficulty. It is anticipated that in the next several years both medical therapy and transplantation will result in significant improvement, with, hopefully, a much more positive outlook toward patients who present with this problem.

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# Life Support Learning Center

R. F. Edlich, MD, B. T. Barbour, EMT,  
E. R. Levesque, EMT, and  
R. F. Edlich, Jr., *Charlottesville*

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Since its inception in 1981, an educational program at a Virginia teaching hospital has offered training in basic and advanced life support to 1,951 health professionals, including many physicians. The program is described to encourage other hospitals to offer similar instruction.

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**A**PPROXIMATELY 20% of resuscitation attempts throughout hospitals (outside of operating rooms and special care units) result in survival with prearrest central nervous system status.<sup>1,2</sup> Salvage rates of over 50% have been reported in operating rooms and special care units after resuscitation of patients.<sup>3</sup> The success of resuscitation during the last 15 years has led to the realization that physicians (regardless of specialty) and nurses should be capable of restoring spontaneous circulation in patients with cardiac arrest and performing various aspects of definitive therapy.

During the past 15 years educational programs have been specially designed using nationally standardized curricula to prepare health professionals to deliver optimal emergency care to their patients with life-threatening illnesses and/or injuries. With the advent of these training programs, many teaching as well as community hospitals are now offering these comprehensive training programs to their staffs. To facilitate the implementation of these courses, a multi-media educational resource center, called the Life Support Learning Center, was estab-

lished at the University of Virginia Medical Center two years ago.

It is the purpose of this article to describe the organization, operation and experience of our learning center to encourage other hospitals in Virginia to establish similar programs.

The Life Support Learning Center is staffed by two full-time health educators who are basic life support instructors certified by the American Heart Association, Virginia Affiliate. They implement the basic life support training courses as well as coordinate advanced emergency care programs. The director of the Center supervises this intensive educational effort to insure that the training center meets the comprehensive needs of the medical center. The activities of the Center are reviewed by the Hospital Resuscitation Committee, which incorporates new standards and treatments into the curricula as improved methods and techniques evolve. Since this committee has the expertise to evaluate these programs effectively, it bears the responsibility for monitoring and evaluating teaching and performance standards. The curricula offered by the Life Support Learning Center include basic cardiac life support, advanced cardiac life support, and advanced trauma life support. Since its inception, 1,951 health professionals have completed training programs. Certification and recertification in basic life support was accomplished in 1,600 cases. Advanced life support training programs were attended by 351 health professionals.

## Basic Life Support

This program prepares health professionals to prevent circulatory or respiratory arrest or insufficiency through prompt recognition and intervention, and to support externally the circulation and/or respiration of a victim of cardiac and/or respiratory arrest through cardiopulmonary resuscitation (CPR). Training in CPR is undertaken in accordance with the standards of the American Heart Association. The most important aspect of basic life support training is manikin practice. Use of these special manikins allows the student to master external compressions, artificial ventilation, and obstructed airways techniques. Commercially available manikins simulate the human and provide realistic responses to the rescuer's resuscitative efforts. The manikins are specifically designed for testing objectively the performance of CPR. A strip chart recorder is incorporated into the manikin; it records the student's volume and frequency of ventilation, depth of external compression in inches and the presence of incorrect hand position.

From the Life Support Learning Center and the Department of Plastic Surgery, University of Virginia. Address correspondence to Dr. Edlich at Box 322, University of Virginia Medical Center, Charlottesville VA 22908.

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It was the opinion of the latest council on CPR and Emergency Cardiac Care<sup>4</sup> that when CPR is performed improperly or inadequately, artificial ventilation and circulation may be ineffective in supporting life. Furthermore, insurance on rigid standards for the performance of CPR can maximize chances for survival and reduce complications. For example, it has been recently demonstrated that the effectiveness of cardiac compression, as measured by arterial blood pressure values and blood flow in large vessels, can be significantly improved by prolongation of the compression time to 60% of the full compression-relaxation cycle.<sup>5</sup> This definitive prolongation of compression is best learned in a supervised manikin practice. External cardiac compression of short duration ("quick-jabbing") is easily recognized by the physician by reviewing the strip chart recording. Compressions of extremely short duration produce a spike-like inscription on the strip chart recording tape that is substantially narrower than that generated by the proper technique. In a recent report, McIntyre et al<sup>6</sup> have shown that incorrect cardiopulmonary resuscitation techniques performed on a recording manikin correlate with pathophysiologic syndromes seen in man in CPR.

The University of Virginia Medical Center requires that physicians complete this basic life support training program annually as one of the prerequisites for yearly appointment to the medical staff. This basic life support teaching program is implemented entirely by our two health educators. Since the inception of the training program, physicians have been very receptive to this educational experience, as evidenced by the responses to a recent questionnaire. In a random sample of 100 physicians who had completed this training course, 95% of the physicians rated this training program as good. They judged the knowledge and skill of the instructors to be good in 95% of the cases. Seventy-nine per cent of the physicians expressed an interest in annual recertification. This expression of a high level of satisfaction with this program taught by health educators was in sharp contrast to the experience of Dalen and his colleagues<sup>7</sup>; they stated that basic life support training of physicians should be done by physicians rather than by nurses or emergency medical technicians since they believe that physicians are not likely to be receptive to instruction by nurses or emergency medical technicians, no matter how competent the training may be. On the basis of our extensive experience, physicians appear to be, like anyone else, receptive to good teachers.

### **Advanced Cardiac Life Support**

Advanced cardiac life support skills are essential for all physicians and nurses working in emergency care and intensive care areas of the hospital. These skills must also be mastered by those who care for seriously ill patients in hospital settings that do not have immediate access to specialists in resuscitation of the patient with advanced life support techniques. In response to these training needs, an advanced cardiac life support course (ACLS) was developed by the American Heart Association to impart knowledge about emergency cardiac care and to provide the opportunity to develop the psychomotor skills necessary to apply this knowledge.<sup>8</sup> Performance tests have been developed to provide a standard against which performance can be evaluated.

This program consists of 1) the basic life support training; 2) the use of adjunctive equipment and special techniques for establishing and maintaining effective ventilation and circulation; 3) the monitoring and recognition of cardiac dysrhythmias; 4) the establishment and maintenance of an intravenous infusion route; 5) the employment of drug therapy in the treatment of the patient with suspected or overt acute infarction, during both cardiac arrest and in the post-arrest phase. The faculty for these comprehensive programs includes physicians, nurses and allied health personnel, as well as the staff from the Life Support Learning Center. In addition, separate educational programs are offered to physicians to prepare them to become ACLS instructors. While the American Heart Association recommends recertification in advanced cardiac life support every two years, Stross<sup>9</sup> provided convincing evidence that physicians should receive yearly recertification in ACLS skills.

### **Advanced Trauma Life Support**

The Advanced Trauma Life Support (ATLS) course was developed by the American College of Surgeons to teach primary care and emergency physicians the advanced life saving techniques and stabilization procedures that are used within the first hour of trauma management.<sup>10</sup> Implementation of the ATLS training program has already resulted in significant improvement in the seriously injured patient's level of primary care as victims of trauma arrive at the definitive care hospital stabilized and in better condition.<sup>10</sup> The program includes lecture presentations, skill demonstrations and skill practicum. Each participating physician is afforded the opportunity to practice life-saving techniques under live and simulated conditions. Recertification in



advanced trauma life support is recommended every four years. Separate educational programs are also offered to physicians which prepare them to become instructors.

A computer program inventories graduates of these training programs, processes information about the educational experiences of all health professionals and the dates for recertification, and also monitors and evaluates the outcomes of resuscitation performed in the hospital. This outcome analysis may identify pitfalls in resuscitation efforts and suggest improved techniques in resuscitation that can be implemented in the training programs.

In the future, we hope to incorporate a computerized interactive learning system into our basic life support training program. Originally developed by the American Heart Association's Emergency Cardiac Care Subcommittee, this system can be used at the student's convenience since no instructor is required. Conceivably, this learning system could reduce our full-time staff's responsibility for basic life support training, allowing them to coordinate and implement more advanced life support training programs.

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## Cellular Immune Response to Metastatic Brain Tumor

Charles A. Whitten, Jr., MD,  
and Jose M. Estevez, MD,  
*Danville, Virginia*

The lung was the primary site in 22 of the 32 cases of metastatic brain tumors reviewed by the authors; of these, none of the eight small-cell carcinomas showed significant lymphocytic infiltration. The other primary sites were compared statistically. The possible causes for the difference in reactivity are discussed.

**L**YMPHOCYTIC INFILTRATE, be it in primary or in metastatic tumors, is generally considered evidence of cellular immune response by the host against the cancer cells. Lack of immune response to the anaplastic small cell (oat cell) carcinoma of the lung in either primary or metastatic sites, has been well documented in the literature.<sup>1,2</sup> The purpose of this survey is to correlate the lymphocytic infiltrates in brain metastases with their histologic type in a small series of cases autopsied over a period of ten years in a community hospital.

#### Materials and Methods

Thirty-two consecutive cases of metastatic brain tumors autopsied over a period of ten years were reviewed. Available sections from the primary tumors and their brain metastases were examined. Each of the cases was classified as to the type of

From the Department of Neurosurgery (Dr. Whitten) and the Department of Pathology (Dr. Estevez), the Memorial Hospital, Danville, Virginia. Address correspondence to Dr. Whitten at 108 Holbrook, Danville VA 24541.

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Table 1. Series of 32 Metastatic Brain Tumors Autopsied Over Ten Years

Histologic Type	Number	Radio- or Chemo- Therapy	Lymphocytic Infiltrate			Group Peritumor			Necrosis	
			A	Intratumor B	C	A	B	C	Mild	Severe
Lung										
Small cell undiff. Ca	8	4	0	0	8	0	0	8	1	7
Large cell undiff. Ca	2	1	1	0	1	1	0	1	0	2
Adenocarcinoma	6	3	3	3	0	1	3	2	1	5
Squamous cell carcinoma	6	2	4	2	0	2	3	1	1	5
Breast adenocarcinoma	4	4	1	0	3	0	2	2	1	3
Thyroid carcinoma	2	0	1	0	1	0	1	1	1	1
Colon carcinoma	2	0	0	1	1	0	2	0	0	2
Pancreas carcinoma	1	1	1	0	0	0	0	1	0	1
Chronic myelogenous leukemia	1	1	0	0	1	0	0	1	0	0
Totals	32	16	11	6	15	4	11	17	5	26

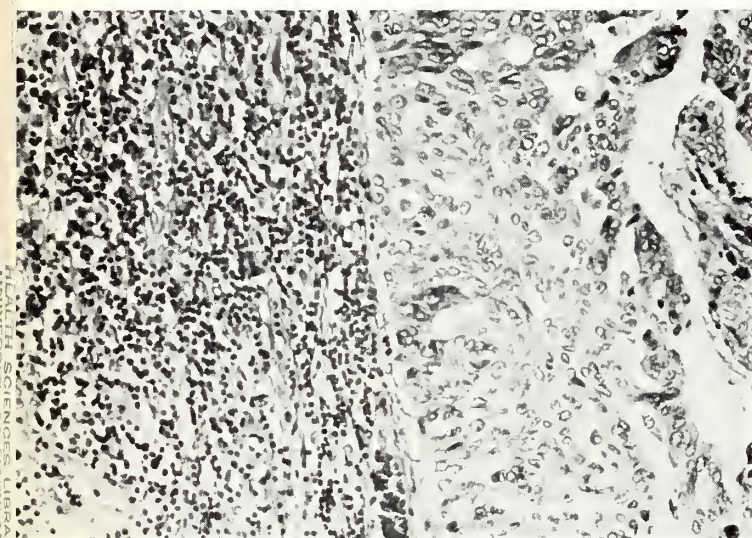


Fig. 1. Abundant intratumor infiltration, lung adenocarcinoma (H&E  $\times$  100).

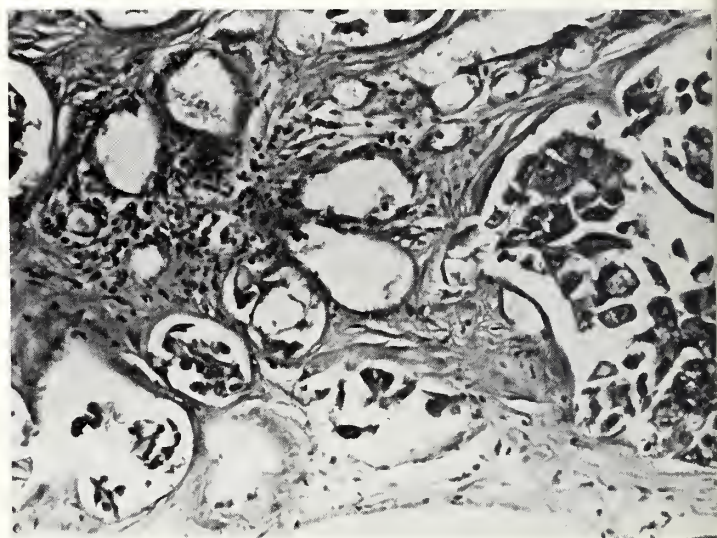


Fig. 3. Mild intratumor infiltration, breast adenocarcinoma (H&E  $\times$  100).

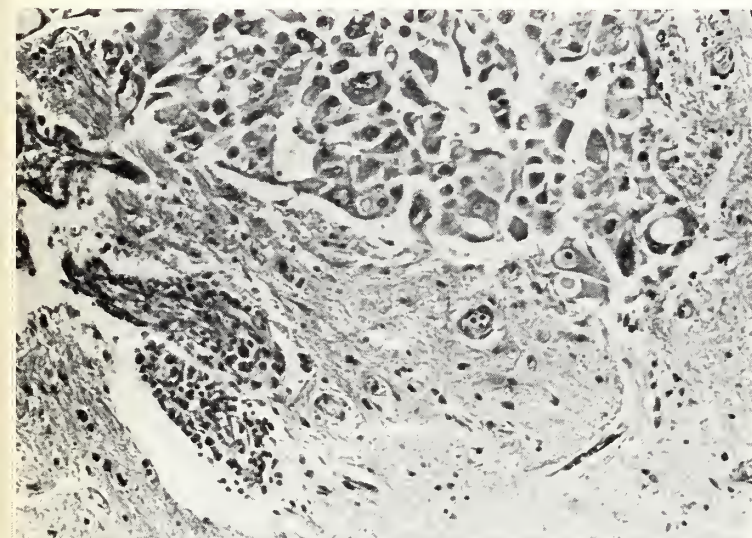


Fig. 2. Peritumor infiltration, breast adenocarcinoma (H&E  $\times$  100).

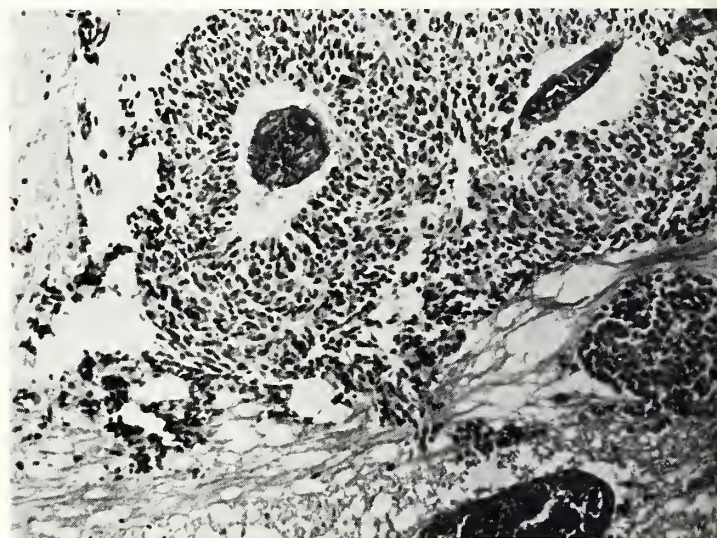


Fig. 4. Oat cell carcinoma, no lymphocytic infiltration (H&E  $\times$  100).



lymphocytic infiltrate (LI) according to the protocol of Palma et al<sup>3</sup> as follows:

Group A (definite): Several perivascular cuffs, with or without parenchymal infiltrates, with more than three layers of lymphocytes. Variable numbers of lymphoid cells, scattered or in groups, infiltrating the tumor stroma. Plasma cells may be found.

Group B (slight): Occasional thin (one or two layers) perivascular lymphocytic cuffs found.

Group C (absent): No lymphoid infiltrates found in any of the sections examined.

In addition, each specimen was graded according to the amount of necrosis from Grade I, absent necrosis, to Grade IV, extensive necrosis with cystic areas.

The type of treatment the patient had undergone during the course of his disease was also noted.

## Results

The histological type of the tumors, their primary sites, the type of lymphocytic infiltrate and the grade of necrosis are noted in Table 1 as well as lymphocytic infiltrate grouping matched to histologic tumor type.

Group A consisted of 11 cases representing most histologic types, except for small cell undifferentiated carcinoma of the lung, and displayed definite perivascular and interstitial lymphocytic infiltrates (Figs. 1 and 2).

Group B included six cases, mostly from the lung, which showed slight interstitial infiltrates (Fig. 3).

Group C, without any infiltration within or around the tumor, included nine cases, eight of which were small cell undifferentiated carcinoma of the lung (Fig. 4).

Histologic necrosis of severe nature was seen in 80% of all tumors. No significant correlation was found between the degree of necrosis and the amount of current antineoplastic therapy, or the degree of lymphocytic infiltrate within or around the metastatic tumor masses. One-half of all the cases had had some type of antineoplastic therapy.

## Discussion

Lymphocytic infiltrates in glial tumors have been extensively studied.<sup>3,4</sup> Correlation between lymphocytic infiltrate and survival has been discussed without conclusive results. We have not been able to find similar studies in metastatic disease to the brain.

Our limited autopsy study precludes conclusions regarding survival, and in most of our cases the brain metastases were incidental findings, having

only a small role in the overall patient's disease process and mechanism of death.

On the other hand, a few general conclusions may be derived from our survey: (a) The immunologically privileged status of the brain does not seem to be supported by the fact that 34% of the cases had significant, definite lymphoid infiltrates. (b) There is no correlation between necrosis and antineoplastic therapy or degree of lymphocytic infiltrate. (c) The histologic type of tumor *does* have a significant bearing in the immune response.

The paucity of immune response to small cell undifferentiated carcinoma of the lung has been discussed for many years.<sup>2</sup> The lack of differentiation of the primary oat cell has been considered the main reason for lack of reactivity: membrane surface antigens are apparently too similar to those of normal cells, or simply synthesis of these specific proteins is significantly impaired, and surface antigens are not deployed, so differentiation from self is not achieved.

Tumors with cells that produce complex proteins such as prekeratin, keratin or mucins seem to be as highly antigenic in the brain as they are elsewhere. Most adenocarcinomas and squamous cell tumors had significant, severe lymphocytic infiltrates.

It has been said that the brain does not harbor lymphoid tissue of its own and that lymphocytes migrate to the nerve tissue directed by opsonic stimuli. The great variability of lymphoid infiltrates to primary and metastatic tumor seem to be more dependent on the type of tumor cell than on the physiological response of the brain itself.

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# What You Can Do About Colorectal Cancer

Wei Li Fang, PhD, Harold Wanebo, MD,  
*Charlottesville, Virginia,*  
and Alvin Zfass, MD, *Richmond, Virginia*

**C**OLORECTAL cancer is the second leading cause of cancer death, surpassed only by lung cancer. In 1984, cancers of the colon and rectum will be responsible for approximately 59,400 deaths in the United States, and an estimated 130,000 new cases will be diagnosed.<sup>1</sup> The majority of these persons will be over the age of 50 since 93% of colorectal cancers are detected in this age group.

Early detection and treatment of cancers of the colon and rectum can lead to an increased five-year survival rate of 75%, compared to the current 44% survival rate. Five-year survival rates have been estimated to be as high as 90% for those who are asymptomatic at the time of diagnosis; 71% for localized disease; 43% for regional involvement; and 8% for distant metastases.<sup>2</sup>

The American Cancer Society and the International Workgroup on Colorectal Cancer recommend the use of three fundamental procedures for the early detection of colorectal cancer in asymptomatic individuals. Two of the three procedures—the digital rectal examination and the stool blood test—are easily employed in a primary care setting. The third procedure—the proctosigmoidoscopic examination—must be conducted by a trained and experienced physician.

In December, 1983, the American Cancer Society targeted colorectal cancer as a Number One priority by launching the nation-wide, three-year Colorectal Health Check Program to increase public and physician awareness of the importance of screening and early detection. Advocacy of the three screening procedures is central to the campaign. A digital

From the University of Virginia School of Medicine (Dr. Fang and Dr. Wanebo) and the Medical College of Virginia/Virginia Commonwealth University (Dr. Zfass). Address correspondence to Dr. Fang at Box 236, University of Virginia Medical Center, Charlottesville VA 22908.

rectal examination is recommended for those age 40 or over on a yearly basis. An annual stool blood test is recommended for those age 50 or over. The proctosigmoidoscope examination is recommended every three to five years after the age of 50, following two normal annual examinations. These guidelines apply only to those persons who have no symptoms.

It is important to recognize that the American Cancer Society guidelines recommend the adoption of all three screening procedures for those age 50 or over. The literature has reported that these techniques should be used to compliment each other.<sup>3-5</sup> If any of the three procedures reveals possible problems, more extensive studies are then necessary. Further diagnostic techniques include barium enema and colonoscopy.<sup>3,5,6</sup>

In the past two decades, a number of studies have been reported whereby screening programs were implemented. Two notable studies were conducted by the University of Minnesota (Colon Cancer Control Study<sup>7</sup>) and the Memorial Sloan-Kettering Cancer Center (Preventive Medicine Institute-Strang Clinic in New York City<sup>8</sup>). Results of both studies have indicated that the rate of positivity for fecal occult blood tests increases for older age groups. Both studies utilized clinical control trials and are tracking patients longitudinally, using experimental and control groups. Currently the studies are in a rescreening and followup stage.

## The Virginia Experience

Under the aegis of the Virginia Division of the American Cancer Society, the Virginia Colorectal Cancer Control Project was established in early 1983. One of the initial steps was the formation of a Steering Committee which was, and continues to be, responsible for developing project goals and objectives and planning activities. Composed of interested physicians, staff of the Virginia Division, and the lay public, the Committee has identified the following objectives: 1) to document the colorectal cancer screening procedures that are currently employed by practicing physicians in Virginia; 2) to increase the awareness of physicians of the value and benefits of early detection and treatment of colorectal cancer; 3) to provide regional educational programs for practicing professionals.

During the summer of 1983, 3,580 physicians in the state received questionnaires which inquired about current screening practices and expressed personal interest in participating in a statewide colorectal cancer screening program. As of October 27, 1983, 800 physicians had responded (22% return



rate), and 600 agreed to participate in the statewide screening project. Of the 800 physicians, 549 (69%) indicated that they regularly screened their asymptomatic adult patients for colorectal cancer. Of these physicians, 581 (73%) tested asymptomatic patients for fecal occult blood by digital rectal exam, and 481 (60%) asked the patients to prepare the Hemoccult® slides at home. Routine proctoscopic exams on asymptomatic patients were performed by 192 of the 800 physicians (24%).

The survey results indicated that Virginia physicians are willing to become involved in a statewide screening project. This led to the establishment of a central registry, where standardized data collection

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See also the editorial on page 577

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forms are sent. These forms seek documentation of the number and characteristics of those screened, types of screening, and diagnostic procedures that were performed. Completed on a monthly basis, data are currently being stored, analyzed, and reported on a periodic basis to both Committee members and to participating physicians. The project provides a method to record the screening of asymptomatic patients by practicing physicians throughout the state. These efforts also emphasize the central role that primary care physicians must provide in a successful screening program.

**W**HAT can you do about colorectal cancer? In asymptomatic patients of average risk, begin routine screening. This consists of an annual digital rectal examination for those age 40 or over and a yearly stool blood test for those age 50 or over. The stool blood test is a three-day test on a meat-free, high-roughage diet for fecal occult blood. In addition, a proctoscopic examination is recommended every three to five years after the age of 50, following two normal annual examinations. In asymptomatic patients of high risk, screening should begin at the age of 20.

In the event of a positive test for fecal occult blood, a diagnostic workup should be initiated; it should include an air-contrast barium enema and a colonoscopy. Most polyps can be removed endoscopically, whereas invasive cancers will require surgical resection.

Become involved with the Colorectal Cancer Control Project. All you have to do is complete and mail to the Registry a single reporting form on each patient who is routinely screened. This information will be compiled regularly, and results will be reported to you.

To encourage continued involvement and to facilitate the recruitment of other physicians, the Virginia Colorectal Cancer Control Project intends to sponsor regional medical education seminars relating to current advances in the screening, diagnosis, and management of colorectal polyps and cancer. Additionally, instruction in flexible fiberoptic proctosigmoidoscopy will be organized in seminars. Participants will have the opportunity to learn the technique on an individual basis. Also planned is a newsletter which will update participants on the progress of the screening project and keep them abreast of new developments and current concepts.

If you have questions about screening and diagnostic procedures, contact the Virginia Division of the American Cancer Society in Richmond (toll-free (800) 552-7996) and your question will be referred to the appropriate source.

To find out more about participation in the Colorectal Cancer Control Project, write to the MCV/UVA Central Registry, Colorectal Cancer Control Project, Box 236, School of Medicine, University of Virginia, Charlottesville, Virginia, 22908.

The authors thank Marcia Nenno and Ethel Sketchley at the Virginia Division, American Cancer Society, for their support and assistance. the Colorectal Cancer Control Project receives financial funds from the Virginia Division.

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# Attention Deficit Disorder Beyond Childhood

William M. Lordi, MD,  
*Richmond, Virginia*

**A**ttention deficit disorder has long been considered a disorder limited to childhood. The purpose of this paper is to extend this concept as it is found in adolescence and adulthood.

In the past, a variety of names have been given to this disorder: hyperkinetic reaction of childhood, hyperkinetic syndrome, hyperactive child syndrome, minimum brain syndrome, minimum brain dysfunction, minimum cerebral dysfunction, minor cerebral dysfunction and many others.

The symptomatology includes inattention with difficulty to finish what one begins and in attending and listening; easy distractibility; difficulty in concentrating on schoolwork and tasks requiring attention, in sticking to play activity and in being interrupted. There is also impulsivity with acting before thinking; shifting excessively from one activity to another; difficulty in organizing one's work and need for much supervision; frequent calling out in class with seemingly inappropriate behavior; difficulty in waiting one's turn in game or group situations. Hyperactivity has been the hallmark of many with attention deficit disorders. Other symptoms are running about, climbing on things, difficulty sitting still, fidgeting, difficulty staying settled, moving about excessively in sleep, and always on the go and driven.

The onset has usually been described before age 7 years. This symptom complex has a genetic and hereditary basis with many precipitating, aggravating factors.

Authors David R. Wood, Frederick Reimherr, Paul Wender,<sup>1</sup> Henry Mann, Stanley Greenspan<sup>2</sup> and others have noted the existence of the symptom complex in adults.

In years gone by, clinical emphasis had been on

Adapted from a speech presented before the 7th World Congress of Psychiatry on July 11, 1983, in Vienna, Austria. Address correspondence to Dr. Lordi at 8635 Mayland Drive, Richmond VA 23229.

the personality disturbance and the behavior, often leading, therefore, to misdiagnosis of such problems as juvenile schizophrenia and pseudo psychopathic schizophrenia. We were essentially in the situation that the ten blind men were in when they examined the elephant, each feeling a different part, each assessing the totality of the organism by the part with which they were in touch. The evidence is mounting that we are dealing essentially with a genetic disturbance based on an error in dopamine metabolism, albeit the precipitating factor may be stress of a social, emotional academic or behavioral nature. One of the apparent reasons for misdiagnosis was the lack of this knowledge. There were a variety of borderline type diagnoses because the personality does indeed resemble in many of its dimensions a "pseudo-borderline" personality configuration.

There may be changes in the symptoms, often with the hyperkinesis becoming hypokinesis if not returning to baseline. There may or may not be associated learning disabilities of a primary nature, so that there may be dyslexia, dyscalculia, dysgraphia; however, this is not a constant feature of attention deficit disorder. It is to be noted that as a child becomes an adolescent and then an adult, personality problems as such may become increasingly maladaptive, often explosive, sometimes violent. These people include those who abuse drugs and alcohol, abuse children or their spouses, sometimes are homicidal or suicidal. There are many who transcend and overcompensate and become quite successful but manifest residual difficulties, such as central processing difficulty in controlling their feelings of anger, fear and other personality problems.

Statistics reveal that approximately 10%-12% of the population has a recognizable degree of attention deficit disorder or one of its many variants. Perhaps half of these reach clinical proportions requiring attention. Interestingly, some of these children present subsequent to minor brain insults.

It is important to note that not only in childhood but in adolescence and adulthood the complete cluster of symptoms do not appear, but rather there are partial clusters. Severity has a high correlation with the intensity and number of symptoms in the clusters.

Obviously, the ideal time to initiate diagnosis and treatment is early childhood, certainly before age 7. Adults, however, are being identified today because they come to the attention of the court, clinician, social agency, lawyer, substance and drug abuse centers. In all fairness, many of the persons with



attention deficit disorder compensate and are remarkable leaders; however, the price they have paid very often is in terms of structural personality problems.

One of the characteristics of the adult patient is difficulty with reality testing and with perceiving what is inner and what is outer influence; judgment suffers because of this, and there is an anticipatory defensiveness. There is frequently derealization, depersonalization. When not under stress, many of these people can process inner and outer stimuli. There is often a primary breakthrough of impulse expression and difficulty expressing feelings. This produces a primitiveness and narcissism in the relationship, and therefore, immaturity. Object constancy is often affected. Memory, concentration, attention, ability to conceptualize and process are impaired, this becoming more apparent under stress.

Medication has been found by many to be extremely efficacious in adults as well as children, which would seem to support a biochemical basis for this disorder. The drug of choice is methylphenidate hydrochloride (Ritalin<sup>®</sup>), with dosages anywhere from 10-20 mgm, which given twice a day often is helpful with adults. The oldest drug, dextro-amphetamine sulphate, has one of the best batting averages. Phenytoin sodium (Dilantin<sup>®</sup>) has been used in the past. Pemoline (Cylert<sup>®</sup>) is popular for treating adults. Imipramine and amitriptyline are gaining in use. Methylphenidate, diphenylhydantoin and methamphetamine are also used.

Medication appears to improve the capacity to attend and therefore to learn, to evaluate, to improve judgmental and cognitive acuity, and to undercut aggression, thus making it possible for more appropriate social, emotional and cognitive behaviors and adjustments.

In addition to medication, it is important to note that psychotherapeutic intervention is most useful. In the beginning, it must have a reality orientation, to be goal limited and symptom-focused. With this working successfully in conjunction with the use of medication and the beginning of increased adaptive behavior, many of these people become able to work at a deeper level in psychoanalytic-oriented psychotherapy, even psychoanalysis. One must work with the family and with the system in which the individual exists. Transference issues usually are not dealt with initially but later on. Long-term psychotherapy is the rule, along with managing the medication. I have had many of these patients on many of the above medications for six to eight years.

Dr. Richard Gardner, in writing about minimal brain dysfunction in children and adolescents,<sup>3</sup> indicated that the four steps of therapy for attention deficit disorder are medication, special education, psychotherapy with the patient, psychotherapy with the family. Treatment has to be attuned to the specific needs and presenting picture. Medication is often indicated, occasionally biofeedback, behavior therapy, educational remediation, correction of general health problems, a change in the environment, family or systems therapy, occupational therapy, and where indicated, in-depth psychotherapy.

Adults usually seek help of their own volition because they are in trouble. The adult requirement is always to perform better on the job and to produce. The major emphasis with children is to learn. Often the maximum treatment some people will permit will be the use of medication and guidance in and around identified problems and interpretations to the family in gaining their support and understanding. There is less concern in the healthy adult about the possibility of weight loss, growth retardation, or problems with cardiovascular system, unless those problems already exist. On the other hand, there is less risk of addiction with children than there is with adults.

In summary, then, attention deficit disorder is a symptom complex that appears to have, in many instances, a genetic hereditary factor with additional precipitating and aggravating factors. The disorder itself is not always accompanied by learning disabilities, but these are frequently concomitants and they appear to be related to some of the similar basic metabolism of neurohormones. There is occasional hyperactivity. Patients may benefit from a combination of medication and psychotherapy consistent with the task at hand and the capacity of the individual. There are problems with the character structure, personality function and cognitive abilities that require attention over and above the symptomatology.

It is felt that as we become more knowledgeable and develop better medication and psychotherapeutic approaches, the prognosis will improve.

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## In a flurry

of hands, Dr. Daniel N. Mohler fields a check for \$13,477 made out to the University of Virginia School of Medicine. Participating in the shuffle are Mrs. Marvin N. Lougheed (left) and Mrs. Rahmat M. Seif, both of The Medical Society of Virginia Auxiliary. The check represented the most recent allotment of AMA-ERF funds to the Charlottesville medical school, where Dr. Mohler is assistant dean. Mrs. Seif is the Auxiliary's president, Mrs. Lougheed is its AMA-ERF chairman. The check was presented to Dr. Mohler at the mid-year meeting of the Auxiliary's board. For the Auxiliary membership, the raising of AMA-ERF funds is a dynamic continuum of Christmas card sales, quilt raffles, portable boutiques, and other money-makers. All three of Virginia's medical schools reap the annual harvest; the Medical College of Virginia is to receive \$15,325 this year and Eastern Virginia Medical School, \$11,785.

The Medical Society of Virginia



contributes to medical education in Virginia, too, through its annual scholarship grants of \$2,000 to each of the state's three medical schools. Shown below are four students who were selected to share in the Society's gift to the University of Virginia; they are listening to Dr. Norman J. Knorr, dean of the School of Medicine. In the front row are Vanessa M. Barnabei, Class of '85, and Scott R. Kerns, Class of '86; behind them are Hilary W. Hoge and Thomas D. Wells, both of the Class of 1984.



Michael Bailey



# VIRGINIA MEDICAL

## Evaluation of Foreign Medical Schools

**I**T is generally perceived that the present supply of physicians in the United States is at least adequate; further it is projected that the next five to ten years will bring an overabundance. In addition to the greatly increased number of graduates of medical schools in the United States, there has been in recent years an ever-increasing number of graduates of foreign medical schools. Many of these are citizens of the United States who have been denied admission to American medical schools. Obviously, this influx of foreign medical graduates compounds the problem.

The oversupply of physicians may not be the major concern. At the very least, certain of the foreign medical schools do not provide adequate instruction, facilities or patient population in the clinical areas. There is presently no need for more physicians in the United States; there will never be a need for inadequately trained physicians.

How can this problem be solved? The Federation of State Medical Boards apparently does not have the authority to accredit or approve foreign medical

schools. As a consequence, each state board of medicine is left to its own devices in this effort.

In Virginia, the Credentials Committee of the State Board of Medicine, ably assisted by Dr. John Molnar of the State Council of Higher Education, has prepared an excellent guideline for the evaluation of foreign medical schools, and hence the graduates of these schools. Each state board will prepare a guideline for that respective state. While some or all of these guidelines may be similar and well-constructed, it is readily apparent that something less than the desired result can be anticipated from this fragmented approach.

A suggestion: the Federation of State Medical Boards should prepare a model for the accreditation of foreign medical schools. Each state board could then adapt the model to its own needs and assume responsibility for accreditation. In that way, some unanimity could be achieved, while individual state control would still exist.

EDWIN L. KENDIG, JR., MD

## To Screen or Not to Screen

**M**OST of us are in the category of "treaters" and only go into action once a disease has become symptomatic. Our role is first that of being a Sherlock Holmes in terms of making a diagnosis, then it is that of advising or applying therapy. It is clearly more logical to prevent a disease by good public advice (e.g., use of seatbelts in autos), but

we have very little information that is helpful in this regard for many diseases and particularly for most human cancers.

An intermediate approach between a focus on prevention and a focus on treatment of symptomatic disease is the detection of "preclinical" or asymptomatic disease, with the hope that the final

outcome will be more successful than with symptomatic disease. In this issue a presentation by Fang and co-authors describes a plan for a statewide colorectal cancer screening effort, and it encourages all of us to get on the screening bandwagon. Since we all know how unsuccessful efforts have been in attempting to reduce mortality from lung cancer and other cancers by a screening approach, it is both logical and appropriate to question the merit of these proposals for colorectal cancer. Is it really worth it?

One approach to answering this question is the use of some simple guidelines that have been proposed by Cadman et al,<sup>1</sup> as follows:

**Has the effectiveness of the screening program been demonstrated in a randomized trial?** Objective scientific evidence of statistically, clinically and socially significant value to screening for any cancer is a tough "requirement". Data are now available that demonstrate the ability of fecal occult blood testing to detect more favorable stage tumors<sup>2</sup>, but it is still not proven that mortality rates are truly altered.

**Are there sufficiently effective treatments for the disorder that will allow us to profit from an early diagnosis?** This guideline may be the Achilles' heel for some cancers (lung, pancreas), but colorectal cancer clearly has efficacious treatment methods. In addition, smaller and lower stage lesions, the kind generally found by screening asymptomatic populations, are the most effectively treated group.

**Does the current problem in our population warrant screening?** The authors emphasize the fact that colorectal cancer is one of the most frequent cancers in our population, and only 50% of symptomatic patients are successfully treated. Presumably,

this result could be improved by screening.

**Is there a good screening test?** The test for occult blood is inexpensive, sensitive, and easy for both physician and patient.

**Does (or can) this screening program reach a high proportion of persons who might be benefited?** That depends on us! The primary physician plays a critical role here.

**Can the health care system cope with the screening program?** Simple screening by testing for fecal occult blood is certainly something our health care system can deal with.

**Will those with a positive screening test comply with the subsequent advice and interventions?** Years ago a screening effort for gastric cancer was marred by refusal of a significant number of patients with abnormal findings to submit to treatment recommendations. Our population has become more sophisticated regarding health matters over the years and this is probably not a major problem now.

How does the proposed statewide colorectal cancer screening program "stack up" in terms of the above criteria? It appears that we still lack scientific proof for a major public health impact from such a program, but there are suggestive factors for major benefit from this approach. If all of us join in with this program here in Virginia, we may well contribute to the scientific proof that this approach is a winner!

WALTER LAWRENCE, JR., MD

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## A Good TIPP

A CAMPAIGN to prevent common injuries in children has been launched by the American Academy of Pediatrics. Called "TIPP," the acronym for The Injury Prevention Program, this effort provides information and materials necessary for physicians to meet the new AAP standard of care which states that anticipatory guidance for injury prevention should be an integral part of medical care and records should reflect that advice. Prevention counsel by physicians has been shown to be effective. Physicians should advise parents to acquire for their children's safety:

1. Approved child car restraints. They are at least

90% effective in preventing deaths in car crashes.<sup>1</sup> Protective seats for all motor car passengers under 4 years of age are now required by law in Virginia. Highest use rates have occurred when active physician support has been coupled with a law.<sup>2</sup>

2. Smoke detectors to protect children's sleeping areas. The death rate in Virginia children from house fires is second only to that from auto crashes. Smoke detectors are effective in reducing deaths in house fires.<sup>3</sup> Physicians' advice to parents recommending the installation of these inexpensive devices is followed.<sup>4,5</sup>

3. Safe hot water temperatures at the tap. Tap



water causes 10%-20% of scalds in children.<sup>6</sup> Water heaters with thermostats set at medium level deliver water at about 150°F, which can produce a full-thickness burn in less than five seconds. Set at the low mark, the water at the tap is about 125°F, reducing the potential for full-thickness burns nearly to zero but yielding sufficient hot water for all of today's household appliances, including dishwashers.

4. Gates or guards at windows and stairways. Falls are one of the most frequent causes of childhood visits to emergency rooms.<sup>7</sup> Death from window falls can be reduced in those populations at risk.<sup>8</sup>

5. One-ounce bottle of syrup of ipecac. This safe, rapid emetic effectively reduces the effect of ingested poisons and is recommended for homes with children under 5 years of age.<sup>9</sup>

To assist physicians with these injury-preventing efforts, the Academy has published "TIPP sheets" which contain up-to-date targeted prevention advice. Press releases have highlighted for the public the need for our involvement.

Physicians can promote injury prevention personally when they see children and parents, who rely on their medical advice and counsel.

LORNE K. GARRETTSON, MD

Box 581, MCV Station  
Richmond VA 23298

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## Medical Society of Virginia Annual Meeting 1984 November 8-10 Williamsburg



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# VIRGINIA MEDICAL OBITUARY

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## **Robert S. Rixse, MD**

Dr. Robert Sheldon Rixse, Alexandria, was fatally shot by an assailant at his home on the night of June 30 and died on July 1 in Alexandria Hospital. He was 37 years old.

Born in the District of Columbia and raised in Alexandria, Dr. Rixse was graduated in 1968 from Johns Hopkins University and in 1972 from Duke University Medical School. He trained at Duke and at Children's Hospital in Washington, DC, before establishing his pediatrics practice in Alexandria.

Dr. Rixse came to membership in The Medical Society of Virginia through the Alexandria Medical Society. He was also a member of the American Academy of Pediatrics.

## **Ray H. Grubbs, MD**

Dr. Ray Huddle Grubbs, Christiansburg general practitioner and surgeon, died at home on February 6. He was 76 years old.

A native of Page County, Dr. Grubbs was graduated from the Emory University School of Medicine, Atlanta, in 1932. He received training in Maryland, Pennsylvania and Kentucky before returning to his home state to establish a practice. He was also staff physician for the Norfolk & Western Railway for over 40 years.

A member of the Southeastern Surgical Congress and the American College of Surgeons, Dr. Grubbs came to membership in The Medical Society of Virginia in 1936, through the Southwestern Virginia Medical Society.

## **Harry B. Stone, Jr., MD**

Dr. Harry Benjamin Stone, Jr., former president of the Roanoke Academy of Medicine, died at age 75 on May 22 in a local hospital. He had practiced ophthalmology and otolaryngology in Roanoke for over 45 years.

A native of Roanoke, Dr. Stone was graduated from Hampden-Sydney College and in 1934 earned his medical degree at the University of Virginia. He received training at the University of Virginia Hos-

pital and at New York Eye and Ear Hospital. From 1942-1946 Dr. Stone served in the Naval Reserve and was stationed in the Panama Canal Zone.

Reared in a medical family, Dr. Stone's father, the late Dr. Harry B. Stone, Sr., preceded him in practice in Roanoke and a brother, Dr. W. Conrad Stone, was his associate, retiring from practice last year. Two sons, Dr. Harry B. Stone III, New Bern, North Carolina, and Dr. Kearfott M. Stone, Gloucester, followed their father in the practice of medicine.

A past president of the Virginia Society of Ophthalmology and Otolaryngology, Dr. Stone was also a member of the American Academy of Ophthalmology and Otolaryngology. His membership in The Medical Society of Virginia spanned 46 years.

## **Memoir of W. O. Bailey 1890-1984**

*By William Otis Bailey, Jr., MD*

Dr. William Otis Bailey died May 19, 1984, in Richmond at the age of 94. Born in Charleston, South Carolina, he moved to Washington at age 9 and was educated in the District of Columbia and Anne Arundel County (Maryland) public schools and at the Emerson Institute. He graduated from the George Washington University with the degree of MD. in 1912, after internship at the old Providence Hospital and further service in the US Marine Hospital in Boston, he entered the private practice of medicine in Washington. Subsequently he became a physician in the Bureau of Indian Affairs and was stationed in Minnesota and Fort Defiance, Arizona.

In 1917 he enlisted in the regular US Navy, attaining the rank of lieutenant-commander. His initial station was Portsmouth, New Hampshire, and he was then, at the age of 28, given command of the medical facilities of the newly acquired US Virgin Islands. He established the first nurses' training school of those islands in Frederiksted, St. Croix, where he and three other young naval medical officers were the only physicians available.

He began the speciality of eye, ear, nose and



throat at the US Naval Hospital at Great Lakes Naval Training Station near Chicago and after that was commanding officer of the Naval Hospital in Key West, Florida. Final service was at the US Naval Medical Center in Washington.

From January 1924 until his retirement at the age of 81 he practiced his speciality of eye, ear, nose and throat in Leesburg, Virginia, and for more than a quarter of a century was the only physician in his speciality in Loudoun and Manassas, Culpeper and Front Royal, Virginia, and Charles Town, West Virginia. In 1928 he took further post-graduate training in his speciality in Vienna, Austria, and Budapest, Hungary. For 25 years he conducted a free clinic in his office each Saturday for Loudoun County welfare cases. A member of Saint James Church in Leesburg, he was superintendant of the Sunday School there for many years. He was a long time secretary of the Loudoun County Medical Society and delegate to The Medical Society of Virginia, a life member of the American Medical Association and the Medical Society of Virginia, and a former member of the Medical Society of the District of Columbia.

His wife died in 1977. He is survived by a daughter, Mrs. Harry L. Merring, Jr., of Scottsdale, Arizona; and two sons, Dr. William O. Bailey, Jr., of Bethesda, Maryland, and the Rev. Edwin P. Bailey of Winnsboro, South Carolina; and seven grandchildren.

## Memoir of M. E. Yeamans 1927-1984

*By Leroy S. McDaniel, MD,  
Randolph McCutcheon, Jr., MD,  
and Lawrence O. Snead, Jr., MD*

Our beloved colleague in the Richmond Academy of Medicine, Melvin Earl Yeamans, died June 3, 1984, having coped with bronchiogenic carcinoma, while continuing active medical practice into December 1983.

He was a native of Richmond, served as a pharmacist's mate in the US Navy during World War II, returned to attend the University of Richmond and continued his studies at the Medical College of Virginia, earning his MD degree in 1952. He served his internship at Norfolk General Hospital and returned to Richmond, where he served for 31 years in general practice, becoming board certified by the American Academy of Family Physicians and serving on committees at both the state and local level.

He was a member of The Medical Society of Virginia and several other professional organizations, serving with distinction twice as president of the Manchester Medical Society.

Dr. Yeamans' activities also included membership in St. James's Episcopal Church, oil painting and the raising of Dorset sheep and Angus cattle. He was a member of the Virginia Angus Association.

His early death is a great loss to the medical profession, to the community and to his patients who respected him both as friend and physician.

The Richmond Academy of Medicine extends deepest sympathy to Dr. Yeamans' wife, Mrs. Doris Lloyd Yeamans, and to their three daughters, Margaret Lee, Elizabeth Louise and Anne Catherine, and to one son, Melvin Earl Yeamans, Jr.

## Memoir of H. L. Riley, Jr. 1903-1984

*By George B. Craddock, MD,  
Porter B. Echols, MD,  
and Josiah E. Haynsworth, MD*

On May 20, 1984, the Lynchburg Academy of Medicine lost one of its most respected members, Dr. Harold L. Riley, Jr., a former president of the Academy. With his death the City of Lynchburg lost an outstanding citizen and his many patients a very able and conscientious physician.

Dr. Riley was born 29 November 1903 in Greenville, South Carolina. He received his grade and high school education in Greenville and his college education at Furman University (then Furman College) and the University of Richmond.

He was graduated from the Department of Medicine of the Medical College of Virginia in 1930, along with two other later presidents of the Lynchburg Academy, Dr. William T. Pugh and Dr. John Wyatt Davis, Jr.

Dr. Riley's post-graduate training and service was at the Johnston-Willis Hospital in Richmond, at the Catawba, Virginia, Tuberculosis Sanatorium and later at the Lynchburg Training School and Hospital in Madison Heights.

Fortunately for so many of us, he moved across the James River and entered private practice in Lynchburg in 1936. He gave many hours of service in the city's medical clinics and as an attending physician for unassigned ("clinic") patients at Lynchburg General Hospital. Dr. Riley also served several terms on the Lynchburg School Board,

acted as physician for the Odd Fellows Home and as college physician for Lynchburg College.

Dr. Riley's family are medically oriented. His wife, Mrs. Josephine Noel Riley, was a nurse at Johnston-Willis Hospital; a son, Harold Riley III, is a practicing neurologist in Lynchburg; two other sons, Bill and David, are dentists in Lynchburg and Rustburg; and a daughter, Pat, is an RN and the wife of Dr. James E. Blackburn, Lynchburg orthopedist. Another daughter, Mary Jo Booth, is a schoolteacher.

Dr. Riley exhibited great courage in meeting his terminal illness, cooperating fully with his physicians. Because he received wonderful support, both physical and emotional, from his devoted wife and the other members of his family, he was able to pass his last days at home.

Dr. Riley was a person we all admired greatly, and all feel richer having known him.

### **Robert L. Gilliam, MD**

Dr. Robert L. Gilliam, Warsaw, died April 16, at age 75. He was graduated from Emory University School of Medicine, Class of 1934, and trained at Clifton Forge-Huntington Hospital, Virginia, and St. Joseph's Infirmary, now St. Joseph's Hospital, Atlanta. He was a member of The Medical Society of Virginia and the Northern Neck Medical Society.

### **Walter C. Elliott, MD**

Dr. Walter Carlton Elliott, Lebanon, died at his home on July 17, at the age of 81. He was a family practitioner and a former delegate to the Virginia General Assembly.

A native of Campbell County, Virginia, Dr. Elliott was graduated from the University of Richmond and earned his medical degree in 1928 at the Medical College of Virginia. He trained at MCV and at Cook County Hospital in Chicago. He practiced in a coal camp prior to establishing a practice in Lebanon, where he subsequently founded Lebanon General Hospital. In 1952 he first won election to the General Assembly, continuing to serve for 12 years.

Dr. Elliott had been a member of the Southwestern Virginia Medical Society and The Medical Society of Virginia since 1932 and belonged also to the American Academy of Family Physicians and the Industrial Medical Association.

A brother, Dr. James W. Elliott, continues to practice in Lebanon.

### **Dean B. Cole, MD**

Dr. Dean Baldwin Cole, Richmond physician and pioneer in the practice of thoracic medicine in Virginia, died on July 11 at the age of 92.

Born in Smyth County, Virginia, Dr. Cole was graduated from the University of Richmond and the Medical College of Virginia, Class of 1917. Following residency at Bellevue Hospital and Saranac Sanatorium in New York, Dr. Cole served in the US Army as a first lieutenant during World War I. Upon his return to Richmond, he began a practice that was to endure for 50 years.

Long a member of The Medical Society of Virginia and the Richmond Academy of Medicine, Dr. Cole's membership in professional organizations included also the American Association for Thoracic Surgery, American College of Physicians and the American Thoracic Society.

### **Memoir of W.T. Green, Jr. 1895-1984**

*By John Rogers Mapp, MD*

Dr. William Thomas Green, Jr., was born in Ashland, Virginia, the son of a Methodist minister from North Carolina and Maria Old of the Driver-Suffolk area. He attended schools in Smithfield, Petersburg, Bedford Academy and Norfolk Academy. He attended college at Randolph Macon. His uncle, Dr. Levi Old of Norfolk, encouraged him to be a physician and he was graduated from the Medical College of Virginia in 1920. He interned at Stuart Circle Hospital, Richmond, and completed his residency in ENT at Bellevue Hospital in New York City. The chief nurse in that department was Elta Moorehead of Findlay, Ohio, and she became his bride in 1923.

The couple settled in Norfolk, where Dr. Green practiced for a time, but with the opening of the Northampton-Accomack Memorial Hospital in 1928, he moved to Nassawadox, where he worked with the hospital's pioneer staff. Dr. Green also had an office in Cape Charles and held clinics in Chincoteague.

Dr. Green became a fellow of the American College of Surgeons in 1930. He served as chairman of the Northampton-Accomack Memorial Hospital staff in 1954. In 1962 the town of Chincoteague cited him for his "many kind deeds." He was recognized by the Virginia Rehabilitation Association for Retarded Children. He was a member of The Medical Society of Virginia for more than 50 years. He was a



faithful Methodist and a talented musician in piano and organ. He gave of his talent freely to churches, weddings and other functions such as the Northampton Nursing School Glee Club and the Hermitage in Onancock.

He retired from practice in 1964 at age 69. He traveled and enjoyed his home. In 1974 he sold his home and took residency in the Hermitage in Onancock, which proved a pleasant haven for his final ten years. He died on April 4 and was laid to rest in Franktown Cemetery beside his beloved wife who preceded him in death in 1971. Survivors include a daughter, who is a psychotherapist in Rochester, New York, a brother, an active farmer, a sister, directress of nursing at Norfolk General Hospital for many years, and three grandsons. Also surviving are Dr. Levi Old, Jr., and Dr. Levi Old III, of Norfolk, his cousins.

## Memoir of Bruce D. Baird 1944-1983

*By Robert E. Berry, MD*

Dr. Bruce Douglas Baird, associate director of surgical education at Roanoke Memorial Hospitals and assistant professor of surgery at the University of Virginia, drowned while on vacation at Emerald Isle, North Carolina, on August 20, 1983. He was 39 years old. His wife, Heidi, and his children, Chardie and Spencer, knew him as a devoted husband and father.

Dr. Baird was born in Butler, Pennsylvania, and graduated from the University of Vermont College of Medicine in 1969. He trained in surgery at the Medical University of South Carolina, which included one year of research as the recipient of the Carl A. Moyer Award from the American Burn Association. After his residency training, he was chief of surgery at the US Naval Hospital at Cherry Point, North Carolina, for three years. Subsequently he joined the teaching faculty at Roanoke Memorial Hospitals.

Dr. Baird became a diplomate of the American

Board of Surgery in 1977 and a fellow of the American College of Surgeons in 1981. He was a member of the American Medical Association, Curtis P. Artz Society, American Society of Parenteral and Enteral Nutrition, Alliance for Continuing Medical Education, American Trauma Society, Virginia Surgical Society, and was a fellow of the Southeastern Surgical Congress. In addition to his teaching responsibilities at Roanoke Memorial Hospitals, Dr. Baird was director of the nutritional support team, clinical coordinator of the trauma program, and served on numerous staff committees. He was particularly interested in traumatic and endocrine surgery, and pioneered parathyroid transplantation in the Roanoke area.

Dr. Baird joined the Roanoke Academy of Medicine and The Medical Society of Virginia in 1978 and was program chairman of the Academy in 1979.

As teacher, physician, administrator, and caring human being, Dr. Baird was an important part of the medical community of Roanoke. He was also an active member of the First Presbyterian Church. He will be remembered as a man of achievement and a man of promise, respected by students, residents, professional colleagues and other members of his hospital family.

## Warren C. Gregory, MD

Dr. Warren Collins Gregory, Winchester, died on December 14, 1983, in Winchester Memorial Hospital. He was 65 years old.

A native of Damascus, Georgia, Dr. Gregory was graduated from Georgia Institute of Technology in 1941 and thereafter joined the Army to serve during World War II. He returned to enter the University of Virginia and in 1950 was awarded his medical degree. He established a pediatrics practice in Winchester, retiring in 1979.

A member of the American Academy of Pediatrics, Dr. Gregory came to membership in The Medical Society of Virginia through the Northern Virginia Medical Society.

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## Malpractice laws a magnet for legislators

Ever since suing doctors for huge sums of money became a national pastime, legislators in the 50 United States have been tinkering with the rules of the game. In Virginia's General Assembly this year there were six attempts to tinker.

A bill that would have revamped the statute of limitations was introduced by Del. William P. Robinson, Jr. Robinson wanted to change the beginning of the two-year limitation period from the date the injury was sustained, as the statute now reads, to the date the injury was discovered.

Introduced by Del. John C. Dicks III (D-Chesterfield) was a bill that would require all malpractice insurance carriers to furnish to the State Insurance Commissioner detailed reports on all closed claims. A similar statute was repealed a couple of years ago.

Offered by Del. C. Hardaway Marks (D-Hopewell) was a bill that would extend Virginia's \$1 million cap on awards to the limits of the provider's insurance coverage, if that figure is greater.

Del. Franklin M. Slayton (D-South Boston) went Marks one better by proposing to do away with the cap altogether.

Slayton also introduced two other bills relating to malpractice. One advocated replacing Virginia's statewide standards of care with a national standard. The other provided for the importation of expert witnesses from anywhere in the country.

All six bills were waylaid.

Robinson's "date of discovery" proposal was killed by the House

Courts of Justice Committee.

Action on the other bills was averted when The Medical Society of Virginia suggested the formation of a joint subcommittee to study Virginia's malpractice system in general and the five bills in particular. The House of Delegates accepted the recommendation, and the bills were thus automatically carried over to the 1985 session.

Here's how the membership of that joint subcommittee shaped up, as appointed by the Speaker of the House and the Senate Committee on Privileges and Elections.

From the General Assembly: Sen. Wiley F. Mitchell, Jr. (R-Alexandria); Delegate Dicks, who had introduced the reporting bill; and Del. Clifton A. Woodrum (D-Roanoke). All are lawyers; they represent both defense and plaintiff's bar.

From the bar: R. Carter Scott III, Richmond, representing the Virginia Bar Association; and John Ward Bane, Hampton, representing the Virginia State Bar.

From medicine: Dr. George M. Nipe, Harrisonburg, ob-gyn specialist and a past president of The Medical Society of Virginia.

From hospital administration: John N. Simpson, Richmond, president of the Virginia Hospital Association.

The subcommittee got into gear with an organizational meeting at which the members elected Woodrum as chairman. He proved "a fine chairman," according to Dr. Nipe, who describes him as "very willing to listen and to understand."

The assembled members then

set about working up a list of items for study. Included were the salient issues of the six waylaid bills, plus malpractice review panels; structured settlements, in which awards are paid out over an extended period; and collateral source rules, under which plaintiff's other sources of compensation are taken into account in a settlement. These last two mechanisms have been legislated in other states but not in Virginia.

From Dr. Nipe came a recommendation that contingency fees would be a good topic for study, but his suggestion was not well received. Later, when Dr. Nipe protested to Chairman Woodrum that as long as all the other recommendations for study topics had been approved, his should have been, too, Woodrum agreed to add contingency fees to the list.

For the hearing on rate-making, an audience of about 50 showed up in a committee room of the General Assembly Building in downtown Richmond. The Medical Society of Virginia was represented in force by legal counsel, the Society's actuary, an economist, and a contingent of staff, including Dr. Kinloch Nelson, who directs the Society's program of risk management seminars.

Dr. Alvin E. Conner was there. Dr. Conner is chairman of The Medical Society of Virginia's Insurance Committee; his discussion of the malpractice system is featured in this issue of VIRGINIA MEDICAL beginning on page 620.

Dr. Ronald K. Davis was there. Dr. Davis is chairman of the ad hoc



# MS BURFALL

study committee appointed by MSV President C. Barrie Cook to keep a weather eye on the subcommittee's proceedings and to testify as appropriate at its hearings. Dr. Conner is a member of that committee. So are Dr. James L. Ghaphery, Richmond; Dr. Claude P. Sherman, Martinsville; Dr. Robert L. Adeson, Alexandria; Dr. Emerson D. Farley, Richmond; Dr. H. George White, Jr., Winchester; and Dr. Harold L. Williams, Newport News.

Representatives of the Virginia Trial Lawyers Association were there. The association operates out of Charlottesville under the direction of W. Roger Adams. VIRGINIA MEDICAL's reporter called Adams to ask how many of the 17,000 lawyers in Virginia belong to the association. About 2,700, Adams replied. And how many of those members are in the General Assembly? Adams said he didn't know. A spokesman for the Virginia State Bar didn't know, either, but did have the figures on how many lawyer-legislators the General Assembly has altogether. Of 40 senators, 19 are lawyers. Of 100 delegates, 49 are lawyers. That's roughly 49%, down substantially from a previous high of 70%.

The Trial Lawyers Association's turn to testify came early in the hearing. As those in the audience recalled afterwards, the association spokesman's chief complaint seemed to be that malpractice insurance carriers are making an awful lot of money. According to one listener, a profit margin of 51% was suggested.

Virginia's Deputy Commissioner of Insurance, whose boss is charged with guarding Virginians against unruly insurance rates,

rose to refute the suggestion of big carrier profits. Support for his point of view came from The Medical Society of Virginia's actuary and from representatives of the Virginia Insurance Reciprocal and of General Reinsurance, the firm that insures insurance companies.

The air began turning blue with actuarial jargon, and from time to time Chairman Woodrum interrupted to ask for help through the labyrinth of imputed economic gain, retrospective total return pricing, reserving, loss and loss expense, and other such esoterica.

The chairman also asked frequently for opinions on the awards cap. What effect would its removal have on premiums? No one offered empirical data one way or another.

A change of pace was provided by Del. Bernard S. Cohen (D-Alexandria), who took the microphone to testify. He was there not as a member of the legislature, he said, but as a citizen, and he launched into remarks about the insurance industry so linguistically colorful that Chairman Woodrum asked him to cool it.

The afternoon wore on without a lunch break. Finally the last testimony was heard, brief cases started snapping shut, and the chairman announced the date of the next hearing. The items scheduled for that hearing: 1) the cap on awards; 2) carrier reporting requirements; and 3) medical malpractice review panels.

These three topics were the target of Dr. Davis' committee when it met shortly after the hearing. Looking toward possible testimony at the second hearing, Dr. Davis parceled out the three topics as study assignments.

The committee is also boning up on legislative action beyond Virginia's boundaries. In Washington, for instance, the Moore-Gephard bill introduced this year would impose on malpractice litigation a version of "no-fault" insurance that would eliminate contingency fees and awards for pain and suffering.

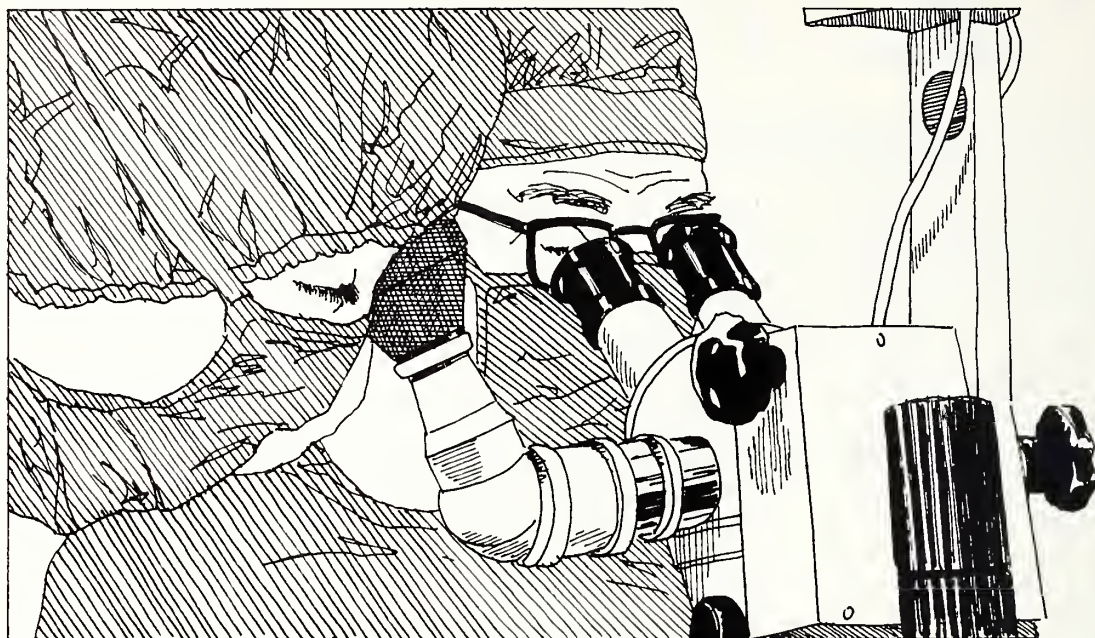
From the office of co-sponsor Rep. W. Henson Moore (R-Louisiana), a spokesman told VIRGINIA MEDICAL's reporter by phone that the bill had little chance of passage this year, but some of its provisions are to be included in a bill to be introduced next year. Moore and Rep. Richard A. Gephard (D-Missouri) also co-sponsored the bill that brought DRGs into being.

The committee would also like to hear someone from the Florida Medical Association talk about the acute malpractice situation in that state. Beset by ballooning injury awards, the association's captive carrier, Florida Physicians Insurance Reciprocal, has seen its patients' compensation fund run dry. As this was written, the Florida Medical Association was petitioning for an amendment to the state constitution that would limit awards for pain and suffering to \$100,000; the amendment would appear on the ballot for referendum in November.

Looking even further, Dr. Davis envisions another role for the committee, one that could give it an ongoing purpose.

"We need to sit down and talk with lawyers from time to time," he told VIRGINIA MEDICAL's reporter. "There's a lot of misunderstanding. Everyone has a point of view. There's a big educational job to do."

—ANN GRAY



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## Congressional Elections '84

**I**N THE CONTEST for the Senate seat, incumbent John Warner (R) is being challenged by former Del. Edythe Harrison (D). Warner has a strong voting record on conservative issues, especially defense, and is well-known across the state. He rates a strong favorite.

Here are the choices in the contests for the ten House seats.

In the 1st district, freshman congressman Herbert Bateman (R) faces the same opponent he faced two years ago, William and Mary Professor John McGlennon (D). Bateman gets high marks for attendance and for his strong conservative stand on the issues. McGlennon has solid blocks of labor and minority votes but not enough name identification and organization. Bateman gets the nod.

Congressman William Whitehurst (R) is unopposed in the 2nd district, and Thomas Bliley (R) is well-liked in the 3rd district and has no major Party opposition. In the 4th district, Norman Sisisky (D) has no major opposition from the Republican Party for reelection. Dan Daniel (D) has held the 5th district seat for years and is unopposed.

Freshman congressman Jim Olin (D) faces determined opposition in the traditionally Republican 6th district. Former State Sen. Ray Garland (R) could win with a strong campaign effort.

After an intense convention nominating process, former Del. French Slaughter (R) emerged to assume the favorite's role over Lewis Costello (D) in the 7th district fight for the seat of retiring congressman Kenneth Robinson.

Contests in the 8th district have always been close and hard fought, and this year is no exception. Incumbent congressman Stan Parris (R) faces a strong challenge from State Sen. Richard Saslaw (D). As this was written, Parris was favored by a small margin.

In the "Fighting Ninth" district, Del. Jeff Stafford (R) is making a determined effort to unseat the incumbent congressman, Frederick Boucher (D). Too close to call, with Boucher a slight favorite.

Like the 8th, the 10th district has perennially close elections, but Frank Wolf (R) has earned the respect of voters by his hard work and energetic attention to the issues and is favored over the challenger, John Flannery (D).

At its meetings in April and June, the VaMPAC board voted to support the campaigns of Warner, Bateman, Garland, Slaughter, Parris, Stafford and Wolf. VaMPAC urges all physicians to support and vote for the candidates of their choice.

**Harold L. Williams, Chairman, VaMPAC**



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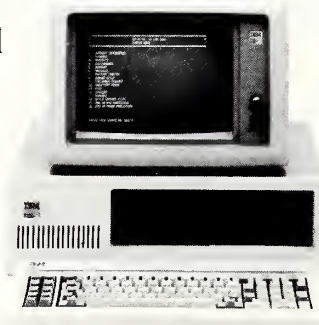
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## From the President: The ACC

**F**ALL is here, but I'm not talking about the ACC basketball teams. I'm speaking of the Annual Communication Catharsis, more commonly referred to as the Annual Meeting of The Medical Society of Virginia.

Who attends and why would anyone want to attend our Annual Meeting?

First of all, as to who traditionally attends, there are the officers, delegates, committee chairmen, and other elected officials. This is an important opportunity for the medical leadership to see old friends and acquaintances and to meet new ones. We communicate and exchange regional problems and ideas for solutions. We participate in the House of Delegates' meetings and reference committees. In addition, we politic a little, or a lot, depending on your bent.

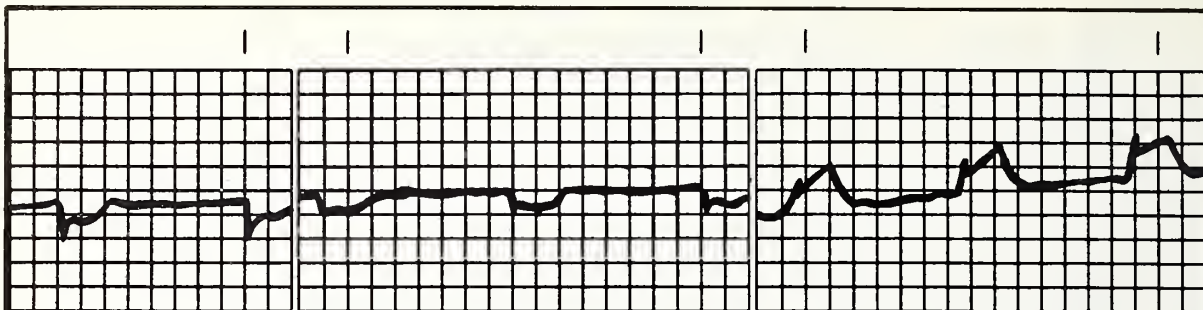
One of the most important events at the Annual Meeting is the reference committee meetings, where considerable discussion takes place on the various resolutions that have been submitted by county medical Societies, committees and, yes, even individual members. After these discussions, sometimes heated, the House of Delegates acts on the resolutions and these resolutions then become part of the policy of The Medical Society of Virginia. This is where a stand is taken on numerous issues that are to come before the State legislature.

At these meetings there is a tremendous outpouring of communication. This is more or less the same format used by the AMA. Some years ago I, as an individual, submitted a resolution concerning blood and its procurement by a pluralistic approach and not by a single entity. This passed our House of Delegates and ultimately went to the AMA and passed there, becoming then AMA policy.

The future health and vitality of our Society depends upon new ideas and the active participation of *all* its members. If there are programs or policies that, in your opinion, the Society ought to be pursuing (or ought not to be pursuing), come to Williamsburg in November and bring your ideas to us through one of the reference committees.

Join the Annual Communication Catharsis. Get involved. Help us continue to make The Medical Society of Virginia responsive to Virginia physicians and the quality health care needs of this Commonwealth.

**C. Barrie Cook, MD**, President  
The Medical Society of Virginia



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**D. Dennis Faludi, MD**, Orthopedics/Hand Surgery, 5021 Seminary Road, Alexandria VA 22311

## *Chesapeake Medical Society*

**Edward S. Hanna, MD**, Family Practice, 515 Albemarle Drive, Chesapeake VA 23320

## *Fairfax County Medical Society*

**Paul A. Buongiorno, MD**, Psychiatry, 3018 Williams Drive, Fairfax VA 22031

**Abraham A. Cherrick, MD**, Physical Medicine/Rehabilitation, 5201 Leesburg Pike, Falls Church VA 22041

**Thomas B. Fleeter, MD**, Orthopedic Surgery, 11355 Sunset Hills Road, Reston VA 22090

**John T. O'Brien, MD**, Cardiovascular Diseases, 313 Park Avenue, Falls Church VA 22046

**James B. Sprague, MD**, Pediatrics, 1515 Chain Bridge Road, McLean VA 22101

## *James River Medical Society*

**Grace E. Suttle, MD**, Internal Medicine, Route 1, Box 416, Palmyra VA 22963

## *Norfolk Academy of Medicine*

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**Richard G. Lester, MD**, Radiology, Eastern Virginia Medical School, Box 1980, Norfolk VA 23501

**Frank H. Kirchner, MD**, Child Psychiatry, 721 Fairfax Avenue, Norfolk VA 23501

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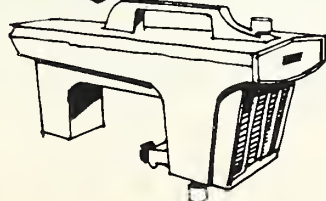
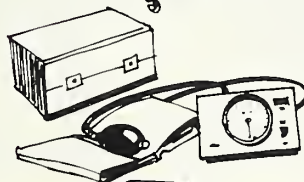
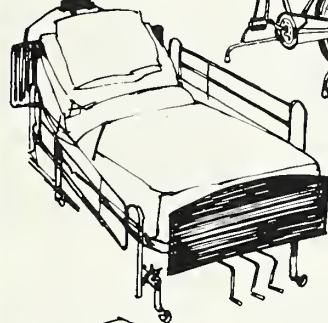
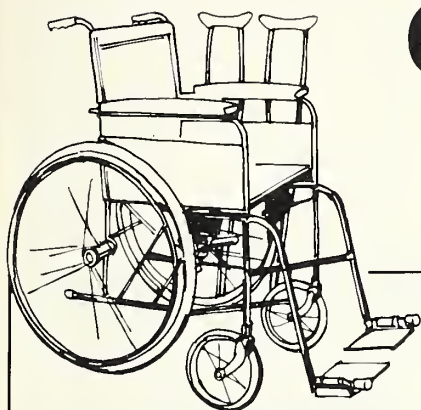
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At a meeting of the Insurance Committee: from left, Dr. Alvin E. Conner, Chairman; Dr. William S. Burton; Dr. J. William Giesen; and Dr. Wilson C. Merchant III.



**W** E PHYSICIANS carry out our life's work in an environment where the product of each delivery is expected to be perfect; where decisions made in crisis must contain the wisdom and judgement that, in other professions, would be made only after lengthy study and deliberation; where lay people determine culpability in situations fuzzy even to the professionals; and where our professional fate may be harmed by a malpractice system operating not from a designed sociological aim but from a mishmash of premises: punishment, compensation, life maintenance, deterrence, common law, statutes, case law, and, finally, from an unfocussed concept of societal responsibility. Because the system has no distinct sociological aim and therefore no inclusive theory to support it conceptually, the wrongs are seldom righted and those in the right are often wronged. Even when the wrong is recognized, the system delivers to the victims approximately 25% of the premiums; 75% is swallowed by the system.

Why did this predicament develop? The answer to this question is elusive. With no great authority, I submit three reasons:

- The consumer movement of the sixties and the litigious society that it produced.
- The over-sell by the medical profession, in which it promised to deliver more than it is capable of delivering, not only to the individual patient but towards the health of the American society.

Dr. Conner has been chairman of The Medical Society of Virginia's Insurance Committee since 1981. He prepared this text for delivery at a seminar on malpractice before the Virginia Obstetrical and Gynecological Society on March 31, 1984, in Alexandria. Address correspondence to him at 8722 Sudley Road, Manassas VA 22110.

**“The wrongs are seldom righted and those in the right are often wronged. Even when the wrong is recognized, 25% goes to the victim, 75% is swallowed by the system.”**

- The growth of economic rewards to participants in the malpractice system, which gives them an incentive to see that the system not only remains the same but also grows in its application.

Whatever the causes of our expensive malpractice coverage, the predicament is real and the solution hard to come by. And so the question: What does The Medical Society of Virginia do to help its members cope with the malpractice aspect of their practice? The answer is short and succinct: the best it can.

**M**EDICAL MALPRACTICE coverage is based in economics. These economic forces yield only to the changing dynamics of the marketplace. The insurance companies sell a product, medical malpractice policies, to make a profit. As long as they make a profit, they will continue to sell their product to us. When we are unwilling to pay a price for their product that ensures profitability or when their costs eliminate any reasonable expectation of future gain, they will get out of the business. This is true both of the commercial carriers and of the

reciprocal. Their only aim is to make a profit, not to serve society.

The Medical Society of Virginia must react within this economic scenario. Therefore, its options and avenues of action are limited by business realities. A group of kindred professionals such as we has only two features that are attractive to an insurance company. First, because we are a homogeneous group under one organizational umbrella, marketing the product to us is easier than to the general population. Endorsement by the Society sells insurance. Second, the group provides an economy of scale that minimizes overhead, and the size of the group spreads the risk. To further increase the basic attractiveness of our business to the insurance companies, we sponsor one product of a kind and concentrate these products in one company. For us, this also increases our leverage during negotiations. It is apparent that The Society will have the leverage to assist in negotiations only if enough of its members subscribe to the endorsed plans.

Because of these two factors, association business is attractive to insurance companies, and they seek us out—but not at any cost. There is a choke point at which our business is no longer attractive because of slim profits. We negotiate, in the interest of our members, to reach this point. What leverage do we have and what are our negotiation aims? We have no leverage if there are no profits. If profitability is possible for the companies, we attempt to secure these: 1) accessibility, 2) the lowest price consistent with profitability. If profits are excellent, we can also demand excellent service.

Of these two business objectives, accessibility to the market is the most important to a collegial association. By this term, we mean that a member of our organization

is able to buy a policy regardless of his personal risk factor. We have attained this feature in our contract with the Blues; otherwise, some of our members would be unable to get hospitalization coverage. St. Paul honors this concept to a great extent, though not bound by agreement. However, the reciprocal companies have an expressed policy of not accepting bad risks, one factor in their ability to offer lower rates.

The Medical Society of Virginia through its Insurance Committee also must consider the factor of availability. We must have malpractice coverage available for our members on a continuing basis. St. Paul, which has provided us coverage for 28 years, is the most likely source for this continuing coverage. Other companies have entered and left the business, depending on economic circumstance. St. Paul has stayed with us, even in the bad times of 1973-1974.

**B**ALANCING these priorities has not been easy. The officers and staff of the Society have worked hard to provide and maintain plans that are accessible to all members with a promise of continuing availability at a price that is reasonable for what we receive. The staff's primary duty is to provide help to members with insurance problems and to provide support for the Society committees that have responsibilities in the malpractice area. There are four of these committees: the Legislative Committee, which represents the Society's interest in matters relating to the General Assembly; the Education Committee, which has jurisdiction over the risk management programs; the Liaison Committee to the State Bar, which, jointly with the State Bar, sponsors the medical malpractice screening panels; and the Insurance Committee, which concerns itself only with

insurance, endorsing programs offered through the David A. Dyer Insurance Agency and negotiating the agreements with St. Paul and the Blues.

The burden of the Insurance Committee has increased drastically in the past several years due to the restructuring of the third-party payer system and to the worsening of the medical malpractice experience. Until that time, committee review and acceptance of figures from the Blues and St. Paul were almost pro forma. But times changed. Competition developed in the malpractice insurance market even in the face of more claims and higher awards. St. Paul, as well as the Committee, wanted to develop an agreement based on a formula that recognized St. Paul's need for an adequate return on capital and, at the same time, assured the physicians that they were not being overcharged. This, then, would provide a sound and equitable basis to continue the St. Paul's program as competition increased. This basis, recognizing St. Paul's needs, would provide the two features of our agreement which are so desirable to association members: avail-

**"We have not raised our placards and marched in the streets because we have been able to pass the costs along by increasing our fees. Most of us have not been hurt at all."**

ability and accessibility.

The formula adopted is contained in the concept of insurance which St. Paul calls retrospective total return pricing. Simply, it is this: St. Paul, from the total income of premiums and interest earned on reserves, gets approximately 12% for profit and taxes and approximately 18% for overhead. The remaining income is used to pay claims. Excess money is returned to the policy holders; shortfalls are absorbed by St. Paul. This differs from the previous arrangement in that interest income from reserves is included in the total income.

The meaning of figures in the insurance industry is difficult to understand. So, to implement this agreement, the Society retains an actuarial firm to monitor our experience through data furnished by St. Paul. Our actuary gives us periodic reports as does St. Paul, providing a basis in fact for Committee decisions. The agreement has not yet been in effect long enough to provide meaningful experience.

**R**ECOGNIZING the efforts of the staff and committees of the Society, these questions must still come to mind: Why don't we do more? Why don't we do something to reduce the number of suits and size of awards? Can't we eliminate the financial and personal threat of malpractice suits? The answer is simple: We are doing all we can.

Malpractice, with all its ramifications, is a societal problem and reform must come about from societal forces, not from the efforts of the medical community alone. Changes in a democracy come about when forces strong enough to effect those changes are benefited. At this time, there is no organized constituency that would benefit from a change in the medical malpractice system. Look at the position of the players in the medical malpractice game. First to mind



are the commercial insurance companies. They are profitable. They may not make much off of the malpractice policies, but these policies bring the additional coverage of physicians' homes and offices, a line of insurance that is profitable. So despite the fact the insurance industry is one of the most potent commercial forces in this nation with the attendant political clout, it has no reason to push for drastic reform. The physician-owned companies have even less reason to seek change. Were the malpractice premiums to decline to the level of 1965, few if any states could develop a premium volume large enough to support a company. This means no jobs for management. We cannot expect help from them.

Next to mind is the legal profession and we certainly cannot expect help from them. Some lawyers make their living on the defense side; others on the plaintiff side. Lawyers not involved give collegial support to those involved; furthermore, the system resolves disputes along lines that lawyers are trained for and approve. Why tinker with a mechanism that is working?

Should the federal government get involved? We hope not. Those of you who have seen the government's use of the insurance bludgeon to keep banks and savings and loan associations in line understand why.

Surely, then, the physicians and hospitals have reason to mount an all-out drive to change the method of settling disputes resulting from damage by the health care system? Not really. We in the provider field have not been sufficiently harmed in our pocketbooks to raise our placards and march in the streets because we have been able to pass the increased costs of malpractice insurance to the consumer by increasing our fees. Most of us have not been hurt at all. The average pediatrician pays approximately

**“Who will make up the constituency that will push through reforms? A consumer-business coalition is most likely because its members are harmed the most.”**

3.2%<sup>1</sup> of his gross in malpractice insurance premiums. A hospital with an operating budget of \$18 million pays \$35,000, less than two nurses' salaries. These are not panic costs; these are very ordinary expenses. In addition, adjustments are occurring. A reported 25% of the obstetricians in Southern Florida have given up deliveries to reduce their malpractice premiums. Obviously, these were marginal producers. This business will go to the remaining 75% who still deliver, thereby increasing their gross and reducing the percentage cost of their malpractice premiums.

What about the consumers? They are the logical ones to press for reform because ultimately they pay the bill. This has not happened because the malpractice cost as a percent of their total medical bill is small; furthermore, it is hidden in their health insurance premiums. They are hardly aware of a problem. Certainly they have not felt its economic sting and, until this happens, they are unlikely to act. Finally, the few consumers who receive the largesse of the system will in no way press for reform.

A SOCIOLOGIST said that an institution that no longer meets the needs of society will disappear. Medical malpractice has been recognized in history almost since recorded time. Various measures in different cultures have been used to punish or to right the wrong. These measures are no longer used, undoubtedly because the society of that day felt they were not appropriate. The tort system has been used to settle malpractice disputes in the United States at least since 1822. It has not gone away as a means of addressing the problem, indicating that society perceives that the system is applicable to our times. To change that perception, a constituency must be developed from people or categories of people who suffer economic harm, and this constituency must be motivated enough to develop the political will to make adjustments in the system.

Who will make up the constituency which will push through reforms? Obviously, a consumer-business coalition is most likely because its members are harmed the most. If premiums rise to a level that jeopardizes the livelihood of the majority of physicians, they will have the incentive to join this coalition. Until this constituency is formed, the present system will muddle through with Band-Aid<sup>TM</sup> remedies and minor readjustments. And The Medical Society of Virginia will try to provide its members with coverage that is available, accessible, and at the best price possible.

1. Kirchner M: Pediatricians: low earnings, high spirits. *Med Econ*, Mar 19, 1984, pp 144-152

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*See also Dr. Connors' committee report on page 620, the insurance brochure inside the cover, and the News Bureau story on page 590.*

# ANNUAL



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## Aging

The Committee met on June 14, 1984, and received a report from Mr. Bruce Reuben, director of finances of the Virginia Hospital Association, on the status of the DRG program in Virginia, who said that although the program is generally going better than expected, several problems exist: proper medical review, correct coding, education of physicians and medical records departments, and "outliers" are only several areas causing concern. Lengths of stay, net revenues, admissions and hospital staffing are dropping, Mr. Reuben said, adding that these reductions could very well affect quality of care since the program is aimed at cost-cutting only. Ambulatory surgery, PPOs, HMOs, IPAs and emergicenters are also affecting this downward trend, he observed, and PROs will rule further cuts.

The Committee was briefed on the legislative joint study committee studying the malpractice laws in Virginia.

**Donald T. Faulkner, MD,**  
Chairman

## AMA Delegates

The 139th Annual Meeting of the American Medical Association was held in June, and six delegates and six alternate delegates from The Medical Society of Virginia attended. In addition, the President, President-Elect, and Speaker of the House met with your delegates to consider 182 resolutions and 73 reports. We are pleased to report that attendance by Virginia physicians was the largest ever in the history of the Society.

A special highlight for Virginia was the reelection of Dr. William S. Hotchkiss to the Board of Trustees. He will serve as Vice Chairman of the Board for the next three years.

The House approved several recommendations of the Board of Trustees contained in a major report with reference to AMA finances and membership. AMA dues will be maintained at current levels in 1985. The Reference Committee cautioned the House, however, that future AMA dues may have to be increased by \$30 in 1986 and an additional \$30 in 1987 in

order to avoid major reductions in AMA programs and activities in those years.

The House also approved a recommendation that state medical associations will be allocated one extra delegate when 75% or more of the state society's members are also members of the AMA.

The dues exempt policy was revised so that this membership category is limited to members who are at least 70 years of age and fully retired or suffering financial hardship and/or disability. Members who are at least 70 years of age and working no more than 20 hours per week pay one-half of regular dues.

The dominant issue at the Annual Meeting was DRGs and their effect on the quality, cost, and availability of care. The House directed the Board to place a high priority on monitoring the system and report back at the 1984 Interim Meeting.

In other related actions, the House voted to continue the AMA's strong and concentrated efforts to seek elimination of the DRG attestation statement that requires physicians to certify primary





At a meeting of the Cancer Committee: Standing at left, Dr. Robert J. Faulconer, Chairman; seated behind him, Dr. Fred T. Given, Jr., (front) and Dr. Arthur B. Frazier. On the steps, seated front, Dr. Walter Lawrence, Jr. (left), and Dr. Robert S. Boyd; standing, first row, Dr. Saul Kay (left) and Dr. J. Shelton Horsley III; back row, Dr. William W. Old III (left) and Dr. Grayson B. Miller, Jr.

## Cancer

Two meetings of the Cancer Committee were held during the year to address a variety of topics of importance to Society members.

The Committee has worked actively toward the preservation of the Virginia Tumor Registry within the Department of Health. The role of the Committee was highly influential in maintaining the visibility and assuring the ongoing support of the Registry by the government of the Commonwealth. To this end the Registry is now the recipient of continued state funding sufficient to bring aboard virtually all remaining larger Virginia hospitals that are awaiting entrance. The end result should mean that at least 70%

and secondary diagnosis and procedures. AMA will explore legislative and regulatory changes to ensure that differences in DRG-based payments to different categories of hospitals (rural and urban) are based on true differences in the costs of providing services by those hospitals rather than on arbitrary geographic criteria.

The House took an aggressive anti-smoking stance, asking the AMA to urge Congress to strengthen warnings on cigarette packages to say that smoking causes cancer of the mouth, larynx, and lung; is a

major cause of heart disease and emphysema; is addictive; and may result in death.

Pertaining to professional liability, the Board of Trustees submitted a report informing the House that a special task force will be established. The task force is charged with coordinating current AMA activities and focusing them on professional liability insurance. A report will be forthcoming at the 1984 Interim Meeting.

**Michael A. Puzak, MD,**  
Chairman



of the acute care hospital beds in Virginia will be covered by the Registry. An editorial elaborating the role of the Registry is in preparation for VIRGINIA MEDICAL at this writing. It will emphasize the value of the Registry's stored data to the practicing physician.

The Virginia Colorectal Screening Program was begun in November 1983. Seven hundred primary care physicians have made a commitment to participate out of a 4000-physician mailing pool.

The Committee will sponsor a program on "New Approaches to Colorectal Cancer" at the annual meeting of the Society in Williamsburg on November 9. The program will emphasize screening and early detection, conservation of function and the improving prognosis for colorectal cancer patients under new forms of therapeutic management.

**Robert J. Faulconer, MD,**  
Chairman

## Child Health

### Recommendations

1. That The Medical Society of Virginia endorse statutory immunity for persons or agencies reporting children suspected of substance abuse.

2. That The Medical Society of Virginia be supportive of continued quality education programs in Virginia schools to include emphasis on alcohol, tobacco and other substance abuse.

3. That the Virginia legal driving age of 16 be continued on a provisional basis, based on a good driving record, and that a permanent license cannot be issued before age 18. Driving records of those between 16 and 18 would be reviewed annually, and more frequently in the instance of a conviction of a driving offense. A one-year suspension

would be automatic for driving under the influence upon completion of a reexamination and testing. Except under certain circumstances, no youth between 16 and 18 would be allowed to drive from 11 PM until 6 AM.

4. That legislation be introduced to increase the legal drinking age to 21 for all types of alcoholic beverages. Such a change in Virginia statute would have a grandfather clause to allow those currently eligible to drink to continue to do so. The Committee wishes to go on record to initiate liaison with law enforcement officials across the state to jointly address the problem of alcohol and substance abuse.

### General

Members of the Child Health Committee continue to be quite concerned about acts of substance

At a meeting of the Child Health Committee: From left, Dr. Jefferson D. Beale, Jr., Chairman; Dr. H. William Fink; Dr. Carolyn M. McCue; Dr. Douglas E. Pierce; Dr. Thomas S. Chalkley; and Dr. James H. Stallings, Jr.



Photographs by Mary Ellen Butler



abuse involving children in Virginia. A spring meeting of the Committee included an excellent presentation by representatives from the Virginia Department of Education, Department of State Police, and the American College of Emergency Physicians, Virginia Chapter.

The Department of State Police sees a great need for continued emphasis on education as well as heightened awareness programs in Virginia concerning the effects of drinking and driving. Law enforcement officials are highly conscious of the dangers and realities of dealing with an individual who knowingly drives after consuming alcohol or other drugs. It is a problem that endangers anyone who drives.

The Virginia Department of Education is concerned about the presumption that all 16-year-olds are mature enough and eligible to secure a driver's license. Public educators have developed curriculum programs on alcohol and drug abuse, and introduced them to students in grades 6, 8 and 10. Greater parental involvement concerning the needs of children and proper discipline seems to be essential in dealing with school age children.

The American College of Emergency Physicians, Virginia Chapter, expressed special interest in the problem of alcohol consumption, particularly as it relates to their treatment of patients in the emergency room who have suffered injury or illness due to excess consumption. It is believed that a closer liaison on the part of physicians with law enforcement officials would be extremely helpful in understanding and jointly addressing these problems. Unfortunately, physicians, educators and law enforcement officials are frequently confronted with the realities of current law and the constitutional rights of individuals. These issues often conflict with well intended

efforts to deal with problems associated with alcohol and drug abuse. Your Committee shares and supports the interests of increased educational activities and awareness programs in these areas. It is felt that heightened disciplinary activity and legislative reform will help to alleviate this significant social problem.

Jefferson D. Beale, Jr, MD,  
Chairman

## Education

The Committee met in Richmond on May 9, 1984, under the chairmanship of Dr. Eugene Temple. Dr. J. Albert Hagy is a new member of the Committee.

*Accredited Organizations (see accompanying list).* Dr. H. Pearce Maccubbin, director of medical education at Winchester Memorial Hospital, has retired and been replaced by Dr. W. Jackson Helm. The Committee thanks Dr. Maccubbin for the excellent way in which he developed and continued the Winchester program.

The Medical Society of Virginia Education Program is due for resurvey in 1984 by the Accreditation Council on Continuing Medical Education (ACCME). No date has been set.

The Potomac Hospital in Woodbridge is due for resurvey this year.

The Prince William Hospital in Manassas has applied for accreditation.

*Compliance with CME Requirement.*

A total of 1,981 members whose last names begin with A through G were due to report in 1983 on their CME activity in calendar years 1980, 1981 and 1982. Ten were dropped for failure to meet the requirement and two were granted extensions.

A total of 1,685 members whose last names begin with H through N were due to report in 1984 on their

## ACCREDITED FOR CATEGORY I CME

The following have been accredited either directly by the Accreditation Council for Continuing Medical Education or by The Medical Society of Virginia's CME Accrediting Committee. Physicians may wish to write to these hospitals and organizations and ask to be placed on mailing lists for announcements about future programs.

### Organizations:

American Cancer Society, Virginia Division  
Eastern Virginia Medical School  
Medical College of Virginia/VCU  
Medical Society of Virginia Scientific Program Committee  
Northern Virginia Consortium for CME  
University of Virginia School of Medicine  
Virginia State Health Department

### Hospitals:

Community Hospital of Roanoke Valley  
Fairfax Hospital Association  
Memorial Hospital, Danville  
Potomac Hospital, Woodbridge  
Riverside Hospital Medical Staff  
Roanoke Memorial Hospital  
St. Luke's Hospital/McGuire Clinic, Richmond  
Virginia Beach General Hospital  
Winchester Memorial Hospital

CME activity for calendar years 1981, 1982 and 1983. Seven with problems will be reviewed by the Membership Committee. As of August 1, eleven had not reported.

All delinquents will be reviewed by the Membership Committee before January 1, 1985.

*Central Registry of Meetings.* This continues to be active but could be great expanded. Those planning CME program are urged to notify the CME office at The Medical Society of Virginia, 4205 Dover Road, Richmond, Virginia 23221, (804) 353-2721, so that their topics and dates can be listed. Many conflicts could be avoided.

*Risk Management/Malpractice Prevention Program.* In association with the St. Paul Fire & Marine Insurance Companies and our

component societies, we have held 22 such programs covering all areas of the state except Southwest Virginia, where two meetings are being developed by the director of continuing education of the University of Virginia School of Medicine, along with the Southwest Virginia Medical Society; 2,249 physicians have attended the sessions held.

Further meetings of specialists in ob-gyn and emergency medicine were considered but were not supported. Similarly, another program in Northern Virginia seemed desirable but was insufficiently subscribed.

The General Assembly in 1984 passed HJR 20 authorizing a joint subcommittee to study Virginia's medical malpractice laws. This committee met on June 5 and August 8, 1984.

We presented our study of the Virginia Medical Malpractice Review Panels, on which we have been working since May 1983, and were asked to find out, if possible, what has happened since the panels were requested or held.

**T. Eugene Temple, Jr., MD,**  
Chairman

## Emergency Medicine

The Emergency Medicine Committee is still in an organizational phase. The six members of the committee were chosen over the past few months. To date, the Committee has been given no specific task to accomplish, and there is, therefore, no activity to report. The idea behind this Committee was that it should act as a liaison between emergency physicians and

The Medical Society of Virginia. Because there are now more than 300 career emergency physicians in Virginia, and because emergency medicine is a specialty in breadth, crossing all specialty lines, we hope to serve as an information source for the Society.

**Peter A. Sim, MD,**  
Chairman

## Highway Safety

### Recommendations

1. That the current form for the physical examinations of school bus drivers be redesigned to include questions about history of mental illness, diabetes, hypertension, epilepsy, previous alcoholism or drug abuse, and use of medications, all of which might affect the ability to drive a bus.

# The Executive Vice President surveys the year

**W**e are raising the roof at Society headquarters! In order to acquire much needed space, the attic area is being renovated to provide an additional 1500 square feet of offices or storage. The majority of the space will be utilized by existing staff. No additional employees are anticipated at the present time.

Our personnel remains at 12. Each employee has administrative responsibilities and is to be complimented on a job well done.

The past 12 months have been a year of major change for the medical profession. In my previous report I mentioned some concerns for 1983-1984, two of which were government regulations of physicians' fees and hospital charges. These concerns are now a reality, with DRGs and Medicare's fee freeze. There is no doubt that the Deficit Reduction Act of 1984 will be a period of continued change and uncertainty for physicians and their patients.

For some good news, turn to page 624 for Dr. Robert Morton's report on the Medical Society of Virginia Review Organization. As he describes, the application to become a peer review organization was judged "responsive" by the Health Care Fi-

nancing Administration, and after some changes in the plan are submitted, we expect the contract to be awarded. Dr. Morton will have later news in a supplemental report to the House.

As described elsewhere in this issue, a joint subcommittee to study Virginia's medical malpractice laws was authorized at the 1984 General Assembly. So that the Society would be fully prepared to testify before this committee, President Cook appointed an ad hoc committee of the Society chaired by Dr. Ronald K. Davis. The Society also has Dr. George M. Nipe as a representative on the legislature's study committee.

When C. Barrie Cook was installed as President last November, he presented to the House his objectives and goals for the coming year. Not only has he accomplished these, but under his leadership the Society has been extremely involved in many endeavors. Membership growth in the Society and AMA was one of his priorities in addition to an effective committee structure. I think you will agree that the committee reports published in this issue are most impressive, as are the following membership figures:



2. The the physical examination of school bus drivers be performed only by a physician, not a nurse practitioner or physician assistant.

3. That 60 days be the period of time during which the physical examinations of school bus drivers be performed, rather than the current 30 days.

4. That a program be set up in school districts to screen randomly those applying to be school bus drivers to pick up such characteristics as alcoholism or drug abuse, which are difficult to detect through physical examination. Because of the logistics, the Committee advocates a pilot program in three or four communities.

5. That side-arms for the seats in all school buses be installed by January 1, 1986, and that larger reflector mirrors be required to obviate a blind spot that exists on school buses.

6. That legislation be drafted to mandate the use of seat belts by all holders of learner's permits in the State of Virginia as well as occupants of the vehicles they are driving. This should in no way be interpreted as lessening our support for enactment of a mandatory seat belt law for Virginia drivers, patterned after the one recently passed in New York.

The Committee also views the use of seat belts in school buses as desirable but of lower priority than the foregoing.

7. That communications be sent to the appropriate police agencies requesting enforcement of the Virginia Child Passenger Restraint Law. Current enforcement, the Committee feels, is less than optimal.

8. That The Medical Society of Virginia endorse, after some minor modification, pedestrian legislation

recommended by the Virginia Department of Transportation Safety as outlined in the report, "The Pedestrian in the Transportation System," by Stokes and Williams.

9. That The Medical Society of Virginia endorse the proposal presented to the Committee by Dr. Harry S. Holcomb III, Nassawadox, that the blood alcohol level for conviction in drunk driving be changed from .15 to .10.

William J. McAveney, MD,  
Chairman

## Hospital Advisory

Matters of common concern to The Medical Society of Virginia and the Virginia Hospital Association continue to increase and intensify with each passing year. As this was written, the Committee had

August 1, 1983	6,211
New Members	428
Deaths	40
Resigned	68
Dropped	42
Net Increase	278
July 31, 1984	6,489
	+ 260 students
	6,749

By classification, the membership includes 5,405 active members; 240 associate members; 17 past presidents; 648 exempt from dues; 179 resident members; and 260 students.

AMA membership is substantially higher than at this time last year. We have exceeded 5,000 members, thus retaining our official representation of six delegates.

Dr. Cook can be proud of his achievements during the past year. It has been a pleasure to work with him, knowing full well his dedication to organized medicine.

Council will have met on four occasions by the time of the annual meeting. The Executive Committee has held three meetings in addition to several

conference calls. The role of an officer, councilor, vice councilor, AMA delegate or alternate delegate is not an easy task. These are physicians that give unselfishly of their time to represent the Society and organized medicine.

The meetings and conferences which require attendance by the President and other Society officers increase each year. Among these are two sessions of the American Medical Association House of Delegates, AMA National Leadership Conference, Conference of State Society Presidents, Congressional Luncheon, annual meetings of our neighboring state medical societies, and component society meetings.

In summary, this report highlights only a small part of your Society activities during the past year. I encourage the entire membership to keep abreast of the ever-changing times we are facing in organized medicine. We must be assured our voices and opinions are heard on important decisions affecting patients, the profession and the future of health care in the nation.

JAMES L. MOORE, JR.,  
Executive Vice President

met twice this year and another meeting was planned. A mere catalogue of the subjects discussed is indicative of our mutual concerns; DRG's, impaired physicians, outpatient "Doc in the Box" Centers, the obnoxious DRG verification statement, professional liability, professional review organizations, and ongoing monitoring of state legislative activities. These weighty items are of course under the constant purview of the officers and council of The Medical Society as well, but we feel our ongoing rapport with the Virginia Hospital Association through this important Committee is a valuable exercise to maintain a strong and well-informed agency in support of the best interests of the consumers of health care in Virginia.

I wish to express my appreciation for the dedicated cooperation of the Committee members: Drs. Robert M. Allen, T. Winston Gouldin, Edwin J. Harvie, Jr., John J. Krueger, and William E. Painter, as well as our fine staff: Jim Moore, Will Osburn, Allen Goolsby and Richard Immel.

**Gerald J. Fisher, MD,**  
Chairman

## Insurance

The Insurance Committee has met twice since the 1983 annual meeting of the Society and plans to meet at least twice more before the November 1984 session.

Effective January 1, 1984, the Insurance Committee elected to transfer sponsorship of the Blue Cross/Blue Shield program to the Roanoke plan. The Insurance Committee reviewed proposals submitted by several carriers before making a decision. The group health insurance coverage offered to the membership in 1984 is characterized as a comprehensive program, which includes an annual de-

ductible followed by co-payments with an out-of-pocket limit for the year.

In reviewing health insurance proposals for the membership, the Committee has become aware of the intensely competitive health insurance market. This market has resulted in the introduction of new concepts in patient cost-sharing to help keep premium increases to a minimum. The Committee will continue to explore options pertaining to benefit changes and underwriting considerations in the future.

The Insurance Committee met with St. Paul in the spring, which included a visit from the president of the Medical Services Division, Joe Nardi. During this session, St. Paul introduced changes in rates for Virginia physicians' and surgeons' professional liability coverage effective July 1, 1984. Premiums increased from 11% to as high as 40%, with the average adjustment being approximately 32%. These adjustments are primarily attributable to the increased frequency and severity in claims, resulting in a higher pure premium cost for Virginia physicians. For years, Virginia medical malpractice experience has been favorable when compared to countrywide results, however, the trend in Virginia now closely parallels that of the rest of the country in certain areas of experience. St. Paul's statistics reveal that approximately 5.4 physicians per 100 had a claim filed against them in 1983 (as opposed to 3.3 per 100 in 1979). Virginia claim severity has also risen 38.9%, from \$11,569 in 1979 to \$16,069 in 1983. The net result is Virginia's pure premium has jumped 147%, from \$349.00 in 1979 to \$863.00 in 1983. "Pure premium" is the average claim cost per doctor that is insured. Other costs, such as taxes, administration, commissions and profits, are added to pure premium to offset the actual rates charged.

The first accounting of the retrospective total return pricing arrangement with St. Paul was analyzed at the end of December 1983. The first annual accounting of the program (initiated in 1981) showed a negative return. It is further anticipated that no return will be available in the second and possibly subsequent years; therefore, the Insurance Committee endorsed the introduction of a new plan effective July 1, 1984. The Dividend Plan will recognize investment income up front in the rating process. Additionally, investment income will be recognized in the dividend calculation and premiums to \$500,000/1.5 million will be used in the calculations.

As in the past, the plan will continue to protect the policyholder by putting a cap on St. Paul's profits and will return a dividend to the policyholder for any unanticipated income generated by the underwriter.

The President of The Medical Society of Virginia has appointed an ad hoc committee to study the medical malpractice laws in Virginia. The purpose of this Committee will be to interface with the legislative subcommittee appointed by the speaker of the House of Delegates to study the medical malpractice laws in the Commonwealth. Your Insurance Committee Chairman is a member of the ad hoc committee. A summary report of the legislative subcommittee's findings will be submitted to the 1985 Session of the General Assembly.

The David A. Dyer agency continues to offer a variety of excellent insurance products to the membership, including several new programs introduced in 1984. Your Committee also continues to work with and coordinate other insurance products offered through Benefit Concepts Group, Inc., and the Dodson Insurance agency. Members of the Society are invited and



encouraged to submit comments to members of the Insurance Committee concerning modification and changes of current and future programs for the membership.

**Alvin E. Conner, MD,**  
Chairman

## Joint Practice

### Recommendation

That The Medical Society of Virginia appoint District Joint Practice Committees and send letters to all component medical societies advising them of this recommendation.

### General

The Joint Practice Committee meets with a similar committee from the Virginia Nurses' Association to work on improving the quality of patient care through the closer cooperation between nurses and physicians in hospitals and all other places where they interact in patient care.

The Committee held two meetings during the year and worked on the present status of physician assistants, pharmacy technicians and nurses on hospital boards. We received information on these and, for future consideration, 18 recommendations from the National Commission on Nursing.

**C. H. Townes, MD,**  
Chairman

## Legislative

### Recommendation

That The Medical Society of Virginia continue the program begun in 1984 of having members attend on a regular basis committee meetings and general sessions of the Virginia General Assembly.

The Committee feels this program is very helpful in the Society's lobbying efforts. Members of the General Assembly are delighted to see their MD constituents and

are happy to hear their views. The Committee feels this program should be expanded in the future.

### General

The Legislative Committee met on February 7, 1984, to review unanticipated new bills that had been introduced at the General Assembly. Positions on these bills and strategies were developed for support or in opposition according to the actions of the House of Delegates at the Society's annual meeting last year.

The Committee met again on March 20, after the General Assembly's session, to take preliminary action on anticipated bills or bills that had been carried over. Presidents of the component medical societies were invited to this meeting, and we had good representation. It is hoped that more of these presidents will attend the next meeting, as it is important to have good communications between the state and local societies.

It is anticipated that the Committee will meet again after the Society's 1984 annual meeting to try to formulate plans to carry out the wishes of the House of Delegates as expressed at the meeting.

Suggestions from the membership concerning any carryover bills will be greatly appreciated and can be sent to me.

**Percy Wootton, MD,**  
Chairman

## Long-Range Planning

The Committee has met on two occasions this year and plans an additional meeting prior to the annual session. This Committee has several important issues under consideration and will submit a supplemental report to the House in November.

**William J. Hagood, Jr., MD,**  
Chairman

## Maternal Health

### Recommendation

1. The Maternal Health Committee recommends that the following resolution concerning nurse midwives that was adopted at the annual meeting in 1983 be reapproved with a recommendation that it be adopted as presented by the Virginia State Board of Medicine:

"Be it resolved that the Virginia State Board of Medicine should license nurse midwives to practice only under the direct supervision of a qualified obstetrician-gynecologist who has full active staff privileges in a JCAH-approved hospital in the same community in which the midwife practices. Should such an association terminate, the license of the midwife should be temporarily suspended until another association with a qualified obstetrician-gynecologist is established."

This resolution was endorsed by The Medical Society of Virginia and recommended to the State Board of Medicine in 1983.

At a joint meeting of the Virginia State Board of Medicine and Nursing on October 27, 1983, the following statement regarding the practice of nurse midwives in the Commonwealth of Virginia was adopted: "The protocols developed jointly by the physician(s) and the nurse practitioner(s) who is a nurse midwife will include a provision to insure that the physician will review all cases on a regular basis, but not less than once a month. The review shall be documented by the physician on the patients' records which must accompany the patient to the hospital if hospitalization becomes necessary. It is in the best interest of all parties that the nurse practitioner(s) have a written agreement with the sponsoring physician(s)."

The Maternal Health Committee does not feel that this protocol is sufficient and recommends that the

original nurse-midwife resolution be endorsed.

2. The Maternal Health Committee also recommends that hospitals develop their own bylaws regulating nurse-midwife privileges and that physician supervision should be required.

3. As requested by the General Assembly, Governor Robb designated May 1984 as Healthy Mothers/Health Babies Month. A contract was awarded to an advertising agency to conduct a media campaign to increase public awareness of the importance of early and adequate prenatal care. Posters and brochures were sent to local health departments and pharmacies for public education. Media spots have been aired on TV. A prenatal Consumer Checklist has been developed by the Virginia Department of Health with the assistance of the Perinatal Services Advisory Council. The checklist will be used in all health departments.

**The Maternal Health Committee** 1) recommends that the Medical Society of Virginia endorse and support this prenatal Consumer Checklist, and 2) recommends that The Medical Society of Virginia send a copy of the checklist to all family practitioners and obstetricians in the State with a cover letter that the checklist is endorsed by the Maternal Health Committee and The Medical Society of Virginia and that additional copies are available from the Department of Health.

4. According to the latest statistics as reported in the 1983 State-wide Perinatal Services Plan developed by the Perinatal Services Advisory Council, approximately 25% of fetuses that died of natural causes weighed over 2500 grams. A protocol will be developed to study these deaths and pertinent records relating to these deaths will be requested from the hospitals by the Virginia Department of Health.

**After the initial study is completed,**

**it is recommended that the House of Delegates urge the Regional Perinatal Centers to continue to investigate natural fetal deaths in their regions.**

#### **General**

An update of maternal and child health activities was given by Dr. Alice Linyear, director of the Bureau of Maternal and Child Health. As mandated by the 1983 General Assembly, the newborn screening program for homocystinuria, maple syrup urine disease and galactosemia was started by the Virginia Department of Health on August 1, 1984. The American College of Obstetricians and Gynecologists recommends that Rh<sub>0</sub>(D) immune globulin be administered prophylactically to Rh-negative mothers at 28 weeks of pregnancy. The Bureau of Maternal Child Health is providing Rh<sub>0</sub>(D) immune globulin to local health departments to be administered to the Rh-negative mothers.

Senate Bill 200 was passed by the legislature. This bill provides for Medicaid coverage to first-time pregnant women.

A subcommittee of the Maternal Health Committee has developed a protocol and reviewed maternal deaths for 1981, 1982, and 1983. This information was very informative and the findings will be submitted for publication to VIRGINIA MEDICAL. They will also be presented at the annual meeting of the Virginia Obstetrical and Gynecological Society.

**Lucien W. Roberts, MD,**  
Chairman

## **Medicine/Business Coalition**

#### **Recommendation**

**That The Medical Society of Virginia endorse the use of medicine/business coalitions to influence legis-**

**lative reform in health care delivery in the Commonwealth.**

#### **General**

The Committee has met three times since the 1983 Session of the House of Delegates. Issues addressed by the Committee concern the proliferation of alternative health care delivery systems (HMO and PPO) and medical malpractice laws in Virginia. Guest speakers have included a chairman of the board and a medical director of a Virginia-based HMO and three speakers invited to discuss the legislative study of medical malpractice laws in Virginia.

There continues to be significant interest in the changes in the delivery of health care in Virginia as influenced by HMOs and PPOs. One year ago there was only one alternative delivery system headquartered in Virginia (Richmond). Today, alternative systems are established not only in Richmond, but in Tidewater and Northern Virginia. The Committee has kept abreast of these changes and has disseminated information to the local levels through the respective coalitions.

The Committee wishes to report that there are ongoing medicine/business coalition groups in Richmond, Norfolk, Newport News, Hampton, Lynchburg and Roanoke. The continued participation of physicians in these local coalitions has helped to enhance the relationship between physicians, management and employees in Virginia. Physicians have helped to impart a very meaningful and necessary perspective to the issue of health care cost containment and have therefore earned the respect of the business community.

Because of the activities of medicine/business coalition groups in Norfolk, significant legislative change has made available greater financial resources for indigent



care for Norfolk area hospitals. Efforts of Richmond area physicians have resulted in the introduction of several medical malpractice reform bills during the 1984 Session of the General Assembly. These accomplishments suggest the effectiveness of continued physician involvement with local business leaders in order to jointly influence legislative change.

**Edward A. Zakaib, MD,**  
Chairman

## Membership

### Recommendation

**That C. Barrie Cook, President of The Medical Society of Virginia, be nominated for honorary active membership.**

Two additional recommendations, one having to do with the creation of a Hospital Medical Staff Section and the second reducing from 70 to 65 the age for eligibility for dues exemption will be presented by the Bylaws Committee.

### General

The Committee met twice during the year and an additional session is planned prior to the annual meeting in November.

The Committee spent considerable time discussing the development of a Hospital Medical Staff Section in The Medical Society of Virginia. By resolution in 1982, the American Medical Association encouraged each state society to establish a hospital medical staff section. The changing medical care environment, trends in hospital structure and management in the 80s, and issues and problems that are unique to medical staffs have all contributed to interest in the section. Such a section will allow hospital staff physicians to have a unified voice in the affairs of organized medicine through a voting member in the House of Delegates. Mem-

bership will be granted to physicians who are members of The Medical Society of Virginia, are active voting members of a hospital medical staff, and have clinical privileges.

Effective January 1, 1984, your Committee began developing the Resident Physicians Section. This section enables interns, residents and fellows to join our Society and enjoy all the membership rights and privileges of active, practicing physicians. It also calls for the establishment of a local resident physician section at each school of medicine in Virginia. Each school section will be entitled to a voting voice in the House of Delegates through two delegates. Recruitment of housestaff physicians to join the Society has resulted in almost 200 new members during 1984. MSV staff has been working with residents at each of the schools of medicine in order to organize their local sections. It is expected that delegates from each medical school will be in attendance at the annual meeting.

The Membership Committee has recommended that the bylaws provision allowing for dues exemption for those physicians who are fully retired and have been a member of the Society for 10 consecutive years be reduced to age 65 (currently age 70). This recommendation is based in part on the fact that a number of physicians each year elect to retire before age 70 and have been members for much longer than 10 years. These same physicians are currently obligated to pay dues until age 70 in order to maintain their membership. Additionally, it reflects the opinion and esteem the Membership Committee has for the senior members of the Society. Such a change will create a more liberal policy than is espoused by the AMA; however, the resulting financial impact on the Society will be minimal.

Membership in The Medical Society of Virginia continued to increase in 1984. The Committee engaged in a number of recruitment activities, which had a favorable impact on total membership. A detailed report of the membership of the Society is included in the report of the Executive Vice President.

**J. Thomas Hulvey, MD,**  
Chairman

## Mental Health

### Recommendations

1. That the Commonwealth of Virginia actively pursue a policy of improving public mental hospitals to meet the standards of the Joint Commission on Accreditation of Hospitals and that The Medical Society of Virginia support budget requests and the General Assembly of Virginia to accomplish this goal.

2. That The Medical Society of Virginia assist the General Assembly of Virginia and any changes in the laws regarding civil commitment of a patient to a private or a public hospital for psychiatric care with a view to the highest quality medical care and adequate funding be provided for the process established by law.

3. That The Medical Society of Virginia continue to encourage and support the expansion and training of child psychiatry at the three medical schools in Virginia and recognize this area of medical specialty as a critically unmet need.

4. That the Commonwealth of Virginia develop plans for the full appropriate licensure of all personnel employed in mental health clinics and hospitals involved in patient care as required for those serving citizens of the Commonwealth on a fee for service basis (social worker, psychologist).

### General

The committee endorsed the recommendations developed by the

Peninsula Medicine/Business Coalition from the Newport News area urging early recognition and treatment of mental disorders, a broad range of levels of care, peer review for cost containment in psychiatry, and wider use of psychiatric consultation in medical surgical settings.

A great concern was a perceived lack of care for the chronically seriously ill mental patient in Virginia because a combination of reduction in psychiatric service in public mental hospitals in the name of community care without the adequate provision of same in the communities of the Commonwealth.

A desire for adequate insurance for the acute and chronically mental ill remains a prime concern. A climate of vigorous effort to avoid insuring for mental illness or what is felt to be a shortsighted and "marketing" strategy by third party payers was discussed. The Committee opposes the exclusion, in law, for an HMO with regard to some mandated coverage for psychiatric illness as is required of other third party payers. The Committee urges the selection of a psychiatrist member of The Medical Society of Virginia to attend study commissions of the General Assembly as they form and develop legislation with regard to public policy and psychiatric disorders and notes the willingness of the Neuropsychiatric Society of Virginia to assist the Society in these efforts.

It remains concerned about confidentiality of patient records, most especially those forwarded in great detail to third party payers to justify continued treatment and those forwarded to the courts in matters of civil and criminal litigation. In the latter circumstance, the Committee is concerned about the proper confidential safekeeping of these records in the courthouses of the State of Virginia and the timely

return of this information from third party payers and courts to the source from which it is obtained.

The Committee is charged with addressing mental health issues and the review of legislation in this area. Comments and suggestions from the general membership are welcomed.

**James Asa Shield, Jr., MD,**  
Chairman

## Negotiations

### Recommendation

**That the Negotiations Committee of The Medical Society of Virginia be terminated by discontinuance as an active committee.**

### General

The Negotiations Committee was established a number of years ago and has not been called on during this time by either the Council of The Medical Society of Virginia or the House of Delegates to perform any service for the Society. The policy guidelines and procedures for the conduct of business by the Negotiations Committee were approved by the House of Delegates in 1980. The Committee feels that the President and/or Council can reactivate the Committee should the occasion arise.

**Levi W. Hulley, Jr., MD,**  
Chairman

## Peer Review

It is no easy task to obtain a contract with the Department of Health and Human Services to be a professional review organization. Congressional intent in enacting the PRO legislation was to allow peer review more flexibility and to build on the successes of the PSRO program while lessening the bureaucratic regulation of the process. Then DRG payment legislation

laid strict programmatic requirements on medical review entities. Bureaucracy immediately responded with more intensive and prescriptive regulations for PSROs, which are being carried into the PRO program. Additionally, budgetary concerns of both Congress and the Administration mean that PROs must operate in an economical manner while pursuing certain mandated objectives of reducing unnecessary hospital admissions and medically unnecessary procedures. The PRO must also pursue objectives designed to assure and improve the quality of care, and that is the primary reason the Medical Society of Virginia Review Organization (MSVRO) came into being.

The initial proposal submitted by MSVRO called for the state organization to subcontract actual review to the existing PSROs, which is allowed under the law but probably is not too practical given the budgetary constraints. The objectives in the initial proposal were insufficiently stated as to the extent of the problems addressed and the methods of problem resolution. Thus the proposal was not acceptable to the Department of Health and Human Services.

With determined and dedicated effort on the part of the MSVRO board of directors and their consultants, the proposal was rewritten. Instead of subcontractors, there will be review regions established throughout the state to preserve the local physician involvement in the peer review process while eliminating some of the management overhead. Analysis of available PSRO data collected in the past two years enabled the documentation of specific utilization and potential quality problems so that the objectives that were developed are meaningful and deserve attention.

The objectives to reduce unnecessary admissions and procedures



will require physicians to obtain preadmission certification of inpatient necessity for those procedures ordinarily done on an outpatient basis and the necessity for certain other procedures that have shown a significant increase in incidence over the past two years. Random sampling review will be used to identify specific physicians and hospitals showing patterns of unnecessary services. The PRO will have the authority to deny payment retrospectively for unnecessary services.

To improve the quality of care, the MSVRO will attempt to reduce the institutional mortality rates that have been observed in patients with diagnoses of diabetes, transient ischemic attacks, appendectomies, cholecystectomies, acute psychoses, depression reactions and acute adjustment reactions. Analysis of patient records falling into these categories may well point out areas where care can and should be improved in medical, surgical, and psychiatric illnesses. In addition, iatrogenic illness related to certain drugs with a narrow range of therapeutic blood levels will be addressed by chart analysis. Specifically, MSVRO will look for instances where the failure to monitor blood levels of digoxin, guinidine, theophyllin, phenytoin and gentamicin contribute to increased morbidity. Postoperative complications will be monitored and studied in depth. Where patterns of inappropriate care are ascertained, corrective action programs will be instituted.

The PRO must also, by random sampling, assure that admissions are medically necessary and that diagnostic information and coding is accurate. Patients transferred from one hospital to another or to a hospital unit that is exempt from DRG payment and patients admitted within seven days of a prior discharge must have their records

reviewed. All permanent pacemaker implantations and so-called "outlier" cases (prolonged or overly expensive stays) are also to be reviewed.

Review will be conducted in specialty hospitals as well as acute care general hospitals. MSVRO will not review long-term care in nursing homes or chronic disease facilities nor will there be review of ambulatory care. MSVRO will cooperate with the Office of the Inspector General in investigation of suspected fraud or abuse and will make review services available to third party payors or purchasers of health care other than the Medicare program.

The 17-member board of directors of the MSVRO collectively represent well over a hundred years of utilization and quality of care assessment. The regional review committees will draw on the physician expertise present within each review region of the state. The board has designated Eugene F. Poutasse, MD, as the medical director of the MSVRO. Dr. Poutasse will work closely with the regional committees, who in turn will be in close touch with their colleagues and the hospitals in their regions.

An enormous amount of work in the development of this statewide plan for quality assurance has been accomplished by the MSVRO board. Freeman Vaughn, executive director of the South Central Peer Review Organization, served as the lead consultant to the board. Barbara Jennings, RN, executive director of the Colonial Virginia Foundation for Medical Care, was designated as an additional consultant to assist in the identification of problem areas and to write the objective statements, including the methodology achieving those objectives. The executive directors of the Shenandoah Professional Standards Review Foundation, Henry

Hunt, and the Northern Virginia Foundation for Medical Care, Gerard Coleman, lent their expertise. In addition, the boards, committees and staffs of all four Virginia PSROs were cooperative and helpful. James L. Moore, Executive Vice President of The Medical Society of Virginia, and his staff were also most helpful and considerate of the board and its consultants.

With the cooperation of the profession, this peer review organization will achieve its goal of assuring that patients receive the best possible care in the most economical fashion consistent with both their needs and the appropriate standards of quality.

**Robert A. Morton, MD,**  
Chairman

## Pharmacy

### Recommendations

**1. The Pharmacy Committee endorses the Medicaid Drug Utilization Review Program and recommends that The Medical Society of Virginia's Executive Committee make provisional appointments to the four regional review committees, including four alternates, and also appoint one physician to serve on the Therapeutic Review Committee, these provisional appointments to be acted on by the House of Delegates.**

The 1984 session of the Virginia General Assembly enacted the Medicaid Drug Utilization Review Program. The sole purpose of this program is to prevent drug-induced illnesses and hospitalization of Medicaid patients. This is not a fraud and abuse program and is not punitive in any way against physicians. Legal immunity will be provided to members of the review committee. Problem cases will be identified by case number only, and committee members will be protected at all times. The information identified is exempt from the

Freedom of Information Act, Federal Privacy Act and PSRO Protection. The committee will meet once a month to review problem cases and attend a one-day regional training program; its members will be paid for mileage and an honoraria for their time.

2. Mindful that the Society's House of Delegates in 1983 adopted a resolution stating "that The Medical Society of Virginia take an active and aggressive position in support of the use of AMA patient medication forms and other patient educational material by the physicians in the State of Virginia," the Pharmacy Committee recommends a voluntary program, rather than a mandatory program along the following guidelines:

**Strongly urge physicians to use the AMA-PMI sheets in their offices to give personally to patients. Solicit physicians to print a box on their prescription blanks that, when checked, would allow the pharmacists to give the patient a AMA-PMI sheet when the prescription is filled. Request that nurse educators at hospitals use the AMA-PMI sheets in hospitals and inform hospital staffs. Form a coalition with the Virginia Pharmaceutical Association to implement a mechanism whereby pharmacists have AMA-PMI forms available for use on a physician's request. Form a coalition with the Virginia Hospital Association to implement a program for the Pharmacy and Therapeutic Committees in hospitals to use the AMA-PMI sheets as the standardized drug information form.**

3. The Pharmacy Committee recommends that The Medical Society of Virginia's House of Delegates adopt the concept of the AMA Prescription Abuse Data Synthesis Program (PAD) and reaffirm its opposition to a triplicate prescription program.

PADS is designed to help state officials identify potential sources

of drug diversion within their jurisdictions through a rapid, economical, non-intrusive and equitable process of data integration and analysis. PADS can facilitate the investigation and intervention processes that follow identification by engaging the support and cooperation of governmental and voluntary sector organizations in a comprehensive diversion control program. The Committee feels that this program will be more effective, and less costly, than the Virginia State Police Triplicate Prescription Program for Schedule II drugs.

#### **General**

The Pharmacy Committee was very active this year. Meetings were held with M. Lee Morse, president, Health Information Designs, Inc., Washington, DC; Paul E. Galanti, executive director, Virginia Pharmaceutical Association; representatives from the Governor's office, the Department of Criminal Justice Services, the Attorney General's office and the Professional Licensing Boards; and Mrs. Bonnie Wilford, AMA Coordinator for the PADS Program.

The Chairman is most grateful for the input from the Committee members, the Society's staff, Will Osburn, and for the challenging leadership of our President, Barrie Cook.

**Gerald C. Burnett, MD,**  
Chairman

### **Physicians' Health and Effectiveness**

#### **Recommendation**

**That the sum of \$12,000 be appropriated for the work of the committee in 1985 and that Council be authorized to appropriate additional money should the necessity arise.**

The requested appropriation is the same as that of 1984. As of August 1st, approximately \$8,000

has been spent and there are commitments for the rest of the year which will probably be around \$2,000. This will mean that we will underspend our budget by \$1,500-\$2,000. It should also be pointed out that \$6,000 of that spent this year was a bill for our slide show, actually produced in 1983, but the bill not received until this fiscal year.

The Committee does not foresee any major expense this year and feels that the requested amount will be adequate. The only possible additional expenditure would be any change in staffing which will be discussed below.

#### **General**

The first year of official operation of the committee, which began on July 1, 1983, has been one of learning for members of the committee and attempts to educate the membership about the program. We have however, investigated a number of reports and in accordance with instructions of the House of Delegates of last year the following statistics are presented.

During the first full year of operation approximately 40 reports were received by the committee and staff of which 33 have had formal investigation. Of that number 12 have either been closed or have been placed on hold because of lack of evidence of impairment or other reasons. Seven physicians have been hospitalized, three at Ridgeview, two at Arlington and two at Peninsular. Three of these remain hospitalized and the other four are now in a process of rehabilitation and monitoring. One physician voluntarily retired while under investigation. One was from out of the state and his care was taken over by another organization. One physician is now hospitalized for evaluation of possible Alzheimers Disease and four have been referred to local psychiatric



management and AA. There are seven cases pending, five which are in initial stages of investigation and two are still under discussion because of difficulty obtaining the necessary information.

One important step which was taken by Mr. Moore this year was the appointment of Ms. Lorraine McGehee to the position of staff assistant, with her duties divided between our Committee and Membership. With the additional time that we will receive from her it is believed that she will be able to take over many of the activities which have in the past fallen to the Chairman or to the members of the committee. It is hoped that contacts will be made through her at staff headquarters rather than through committee members. With this regard the committee has discussed a medical director and we believe that this is one or two years off, but must be kept in mind if the program goes as rapidly as it has in some other states.

The Committee continues to be concerned about its relationship to the State mandatory reporting law and to the Board of Medicine. The Chairman of the Committee and Ms. McGehee meet with members of the State Board and attorneys in an attempt to iron out some of the problems and hopefully we now have a complete understanding with each other. At the last meeting of the committee it was pointed out that in some other states with the mandatory reporting law the program has had to be discontinued. There will be a workshop on this problem at the annual AMA Conference on Chemical Abuse later this fall, which will be attended by members of our Committee. It is interesting to note that Attorney-General Gerald Balisle's statement elsewhere in this issue indicates a very cooperative posture on his part as far as medicine and chemical abuse are concerned, and hope-

fully this will be helpful in the future operation of the Committee.

The Chairman wishes to thank the members of the Committee and staff and particularly those dedicated physicians who have served as investigators and intervenors throughout the state. There has not been a single occasion this past year in which a member of the Society has declined to serve, and in all cases they have performed their assigned functions promptly and efficiently. It is the grass roots work in the local societies that will make this program successful.

**William H. Barney, MD,**  
Chairman

## Public Relations

Again the 1983 annual meeting of the Medical Society of Virginia was very adequately covered by the media through the excellent efforts of our public relations consultants.

There was no action taken on any matters by this Committee during the past year; therefore, there are no recommendations to be brought before the House of Delegates.

We look forward to even more media coverage this year in Williamsburg due to its ease of accessibility from the major metropolitan areas.

**Frederick K. McCune, MD,**  
Chairman

## Rehabilitation

During the current year the Committee, individually and collectively, has continued its primary role of providing advice and recommendations to the Virginia Department of Rehabilitative Services in regard to medical policies and procedures and appropriate fees for medical services provided the clients of the Department. This input has been of

great value in assisting the Department in providing services to the handicapped citizens of Virginia.

The Committee met on May 20, 1984. Eleven members were in attendance. Also present were nine members of the staff of the Department of Rehabilitative Services. The Committee heard reports on the current status of funding and expansion of services for the Department as a whole and a recent study of problems that had developed at Woodrow Wilson Rehabilitation Center. There was also a report on a study of the Department by the State Department of Management Analysis and Systems Development, with particular emphasis on the recommendation regarding "medical only" physical restorative services. After some discussion, the Committee approved a motion that it go on record supporting the provision of "medical only" services to those handicapped individuals who had a vocational objective and no other available source of funding.

The Committee approved a revised surgical fee schedule. It offered suggestions on how the escalating cost of medical evaluations might be contained. The Committee considered several specific therapeutic procedures and made recommendations to the Department.

**Alexander McCausland, MD,**  
Chairman

## Rural Health

The purpose of this committee is to work closely with the National Health Service Corps and the Virginia Council on Health and Medical Care, coordinating their activities with The Medical Society of Virginia. A joint meeting of the Corps, the Council, and the Rural Health Committee is to be held before The Medical Society of Vir-

ginia's annual meeting to present the year's activities and formulate recommendations for the Society's consideration.

It was the pleasure of the Committee Chairman to attend the 1984 Virginia State 4-H Congress in Blacksburg in June. Our support of this fine organization is well founded, as the proceedings restored faith in the youth of America. By personally placing the gold medal around the neck of the winner of the Health Section of 4-H, at the Donner banquet, my interest in this group's standards of perfection was confirmed. It is strongly recommended that our support and this particular personal involvement be continued by this Committee.

**James L. Patterson, Jr., MD,**  
Chairman

## Scholarship

The Scholarship Committee met twice during the year to discuss The Medical Society of Virginia's current scholarship program and the American Medical Association-Educational Research Foundation and to review a proposal to create a foundation for the purpose of awarding scholarships to Virginians attending medical school.

The Medical Society of Virginia has been represented at the awards day ceremony at each of the three schools of medicine for the past several years. This representation has enhanced the visibility of The Medical Society of Virginia and has generated many favorable comments concerning The Medical Society's scholarship awards (currently \$2,000 per year to each of the three schools of medicine). Participation in these programs will continue in the future.

Members of the Committee are quite concerned about the amount of scholarship and loan funds avail-

able to students going to medical school in this country. A significant amount of loan money has been made available in years past through the medical student guaranteed loan program of AMA-ERF. Regrettably, the economy has prevented further loans under this program. Significantly higher interest rates make commercial and federal loans somewhat prohibitive.

The Scholarship Committee has referred to the Executive Committee a proposal outlining the creation of a foundation operated under the auspices of The Medical Society of Virginia to make loans available to Virginians going to medical school. It is hoped that this proposal will receive favorable consideration and be forwarded to Council for further review. Should a favorable recommendation emanate from both of these committees, a supplemental report to the House of Delegates will be developed prior to the annual meeting.

**Anthony J. Munoz, MD,**  
Chairman

## Sports Medicine

### Recommendations

**1. Expand the athletic trainer education process in conjunction with the Virginia State Health Regulatory Board, the costs of this program to be borne by the registration fees of the athletic trainers.**

**2. Support the Sports Medicine Committee's conclusions concerning the physical therapy issue.**

**3. The Sports Medicine Committee would like to bring to the attention of all Society members the public's changing perception of fitness, wellness, exercise and sports. All members are more likely to become involved in the implementation of fitness programs, the treatment of injuries, and the need for prevention and rehabilitation. It is recommend-**

**ed that Society members proceed promptly when indicated with educational programs designed to deal with these changing fitness and injury patterns.**

### General

The activities of the Sports Medicine Committee have increased dramatically in the past year. The Committee has participated in the following:

1. The process of certification of athletic trainers. This has included dialogue with the Virginia Health Regulatory Agency and the Virginia Athletic Trainers Association. It appears that the final solution will include the strengthening of the Society's previously adopted certification program.

2. The Virginia Physical Therapy Association had legislation introduced tangentially by the Board of Medicine which, in effect, would have allowed independent practice by physical therapists. This measure was defeated by the Senate Health Committee after passing in the House of Delegates. Action by individual Sports Medicine Committee members was instrumental in this appropriate Senate response.

The Sports Medicine Committee subsequently met with the president of the Virginia Physical Therapy Association to discuss various mutual points of interest. It was clear from this meeting that an undetermined percentage of physical therapists are striving for independent practices. It was also clear that an undetermined percentage of therapists do not recognize important deficiencies in their skills which would make this responsibility inappropriate.

The therapists also have concern about potential abuses of physician-controlled physical therapy practice. This concern resulted in occasional press releases impugning the integrity of physicians.



These issues may unfortunately be a continuing source of friction. In the view of the Sports Medicine Committee, physical therapists offer important services in the area of rehabilitation, provided the present diagnostic support and direction of physicians are intact. It is therefore important to maintain an open dialogue to resolve these areas of friction and achieve the appropriate goal of enhanced patient care. In this regard, the Committee has concluded it is appropriate to implement the following:

- Accept and review grievances registered by physical therapists concerning abuses of physician controlled physical therapy practice.

- Accept and review evidences of potential illegal individual practice by physical therapists (including diagnosis and diagnostic testing, such as EMG, interpretation of x-ray, blood tests, etc.).

- Continue with open dialogue to amicably resolve the legitimate concerns of physical therapists.

- Oppose any legislation which allows individual practice of physical therapy.

3. The Committee continues to support the Virginia High School Coaches Association with a sports medicine conference at the coaches' annual meeting. This year's conference was directed by Dr. Frank McCue, with support from Joseph Gieck, RPT, PhD, head athletic trainer at the University of Virginia.

It is with pleasure we note that Mr. McCue has received the initial award for sports medicine from the Coaches Association.

**Robert P. Nirschl, MD,**  
Chairman

## State Bar Liaison

The Committee met in the spring with representatives from the Vir-

ginia State Bar. Discussed was: 1) House Joint Resolution 20; 2) reciprocal programs at annual meetings of the Virginia State Bar and The Medical Society of Virginia; 3) revision of "Principals of Cooperation for Physicians and Attorneys"; and 4) review the results of the medical malpractice screening panels in Virginia.

Representatives from the Virginia State Bar announced their availability to develop programs for the annual meetings of The Medical Society of Virginia and the Virginia State Bar. It was agreed that such programs would be worthwhile and would help to ensure a better understanding between physicians and attorneys. The Medical Society of Virginia will explore the possibility of having members of the State Bar involved in the annual meeting in November.

The Committee discussed at some length The Medical Society of Virginia's interest and probable involvement in the legislative study of medical malpractice laws in Virginia. House Joint Resolution 20 was passed during the 1984 Session of the General Assembly and created a seven-member joint subcommittee to study the medical malpractice laws in the Commonwealth. Physicians and attorneys both share significant interest in the statutes as they are now written and it is felt that the joint subcommittee study will help to provide greater insight to the existing laws.

Both physicians and attorneys have expressed appreciation and acceptance of the "Principals of Cooperation for Physicians and Attorneys in the Commonwealth of Virginia." This publication, which was published jointly by attorneys and physicians, has been used extensively, and the Committee will revise and reprint the brochure later this year.

The executive secretary of the Virginia Supreme Court reports

that 80%-85% of the medical malpractice review panels render a decision favorable to the health care provider. The medical malpractice review panels continue to be requested frequently. The Medical Society of Virginia has initiated a study of the panel process and plans to publish its findings before the end of the year.

**Claude P. Sherman, MD,**  
Chairman

## VaMPAC

The Virginia Medical Political Action Committee continues to improve its membership as it nears its 21st year as the political arm of medicine. Auxiliary memberships and "PAC 250" memberships are at an all-time high.

VaMPAC provides many services to good government. In addition to direct financial assistance and encouragement, the PAC provided valuable political expertise as well as special assistance to warranted campaigns where the medical community's personal involvement could make the difference between winning and losing.

1984 was the second in the series of a three-year joint fund-raising activity by AMPAC/VaMPAC in an effort to involve more of the medical community in the political process and candidate support.

In January, VaMPAC held its second much-acclaimed reception at the Commonwealth Club the evening before the start of the 1984 legislative session to honor its leadership. Also in attendance were "PAC 250" members and spouses, Gov. Charles S. Robb, Lt. Gov. Richard J. Davis and Attorney General Gerald L. Baliles.

The executive director of VaMPAC traveled throughout the state attending many component society meetings, sharing political information and ideas, and encouraging

At a meeting of VIRGINIA MEDICAL'S Editorial Board: Front row, from left, Dr. Edwin L. Kendig, Jr., Editor; Dr. Armistead P. Booker, Dr. Duncan S. Owen, Jr., and Dr. Charles E. Davis, Jr., Associate Editors; and Dr. L. Benjamin Sheppard. Second row, l to r, Dr. Harry W. Easterly III; Dr. Robert Edgar Mitchell, Jr.; Dr. Henry S. Campell, Dr. Raymond S. Brown; and Dr. Walter Lawrence, Jr. Third row, l to r, Dr. James N. Cooper; Dr. Richard S. Crampton; and Dr. Glenn H. Shepard.





grass roots support in the political process.

Eighty-nine percent of candidates supported by the PAC in the last General Assembly election cycle were victorious. VaMPAC supported 93 candidates (47 Democrats and 46 Republicans). VaMPAC/AMPAC are currently working to help our friends during their congressional and senatorial campaign year. This is a record of accomplishment of which the entire medical community can be proud. Yet we should take greater pride in what these facts and statistics actually represent—thousands of doctors and their families who have voluntarily supported the PAC with dollars as well as their valuable time. These are the people who recognize that the future of our health care delivery system is in large measure determined by today's legislative decisions here in Virginia and in Washington DC.

**Harold L. Williams, MD,**  
Chairman

## Vanguard

Following the enactment of the Social Security Reform Legislation in March, 1983, this year saw the institution of a new system of Medicare payments based on diagnosis rather than some percentage of the usual and customary charge. The Vanguard Committee has felt that this year should be one of education not only of physicians but of the public in general concerning the mechanisms, problems and possible consequences on the quality of health care that this system has imposed. To that end our pamphlet, "What Doctors Should Know About DRGs", received wide distribution not only at our annual meeting last year but thru VIRGINIA MEDICAL. Over 15,000 copies were distributed.

Individual members of the Van-

guard Committee during this year have been active in making presentations concerning DRGs not only to their local hospital staffs but also to auxiliary groups (medical), civic clubs, public affairs programs on local television and information given to the media. The purpose of these presentations has been to educate the public concerning this new payment mechanism and what influence it may have on the delivery of medical care.

In the recently concluded session of Congress, acceptance of Medicare assignment and a freeze on Medicare charges was enacted. The Vanguard Committee thanks the many members of the Society who participated in correspondence with their congressmen in an effort to defeat the Rostenkowski amendment and its punitive language tying staff appointment to acceptance of assignment. Although this portion of the legislation was defeated, it is thought that it will appear in the next session of Congress and we will be calling on you again. In our July meeting the Committee studied the Gephardt-Kennedy Bill, the latest proposal for national health insurance, and the Heinz Bill, both of which will be widely discussed in the current session of Congress. At the annual meeting we will be offering additional information to the membership concerning these important pieces of legislation and suggesting ways in which The Medical Society of Virginia may have some influence on enactment of these critical laws.

The third matter of great concern has to do with the federal government's plan to unify Part A and Part B of Medicare into a single payment plan. This will be discussed at our next meeting, and the Vanguard Committee voices its strong opposition to such a plan.

It has been a pleasure to serve as Chairman of this Committee with

its excellent membership and we look forward to another productive year.

**J. H. Hollingsworth, MD,**  
Chairman

## VIRGINIA MEDICAL

Essentially, this same material has been presented as an editorial and the reason is simple: we want to use every avenue (including this committee report) to ensure that the membership is apprised of the present goal of the VIRGINIA MEDICAL.

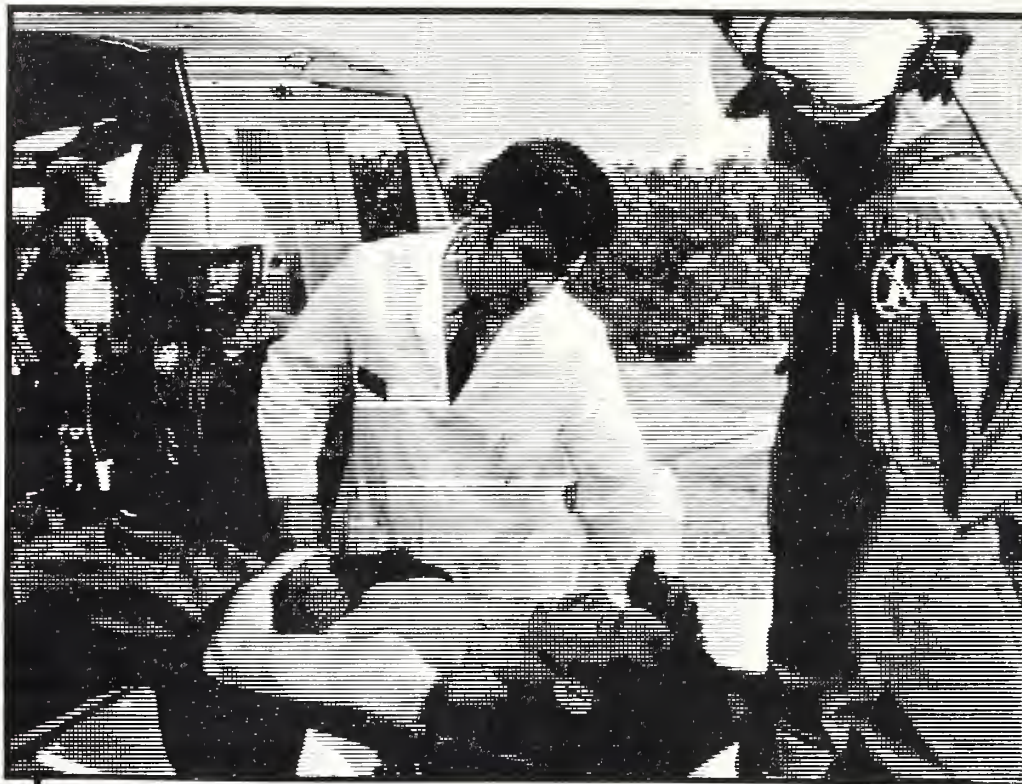
What is the goal of VIRGINIA MEDICAL? Should the journal be a vehicle primarily for the publication of scientific medical articles? Or a promotional organ for The Medical Society of Virginia? Should the space be devoted to the business of the Medical Society of Virginia? Should it be a tool for medical politics? How about a journal for the dispensing of general information as to the activities, both individual and collective, of the medical profession in Virginia?

We try to provide a forum for the publication of scientific articles, primarily by and for the physicians in Virginia. But this is only a part of the story. A considerable portion of the space in the journal is dedicated to the news and activities of medicine in Virginia, an area so beautifully orchestrated by our executive editor, Mrs. Gray. This includes medical politics and state and national politics that affect medicine and overlaps into the third area, i.e., the work of The Medical Society of Virginia.

The goal of VIRGINIA MEDICAL is a blend, consisting of relatively equal parts of scientific articles, Medical Society of Virginia work and general medical news. We hope you like it this way.

**Edwin L. Kendig, Jr., MD,**  
Editor

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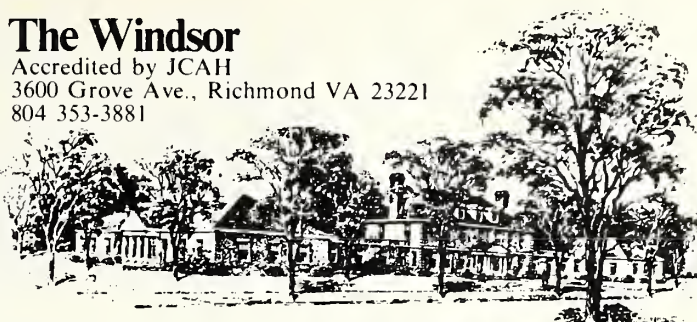




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# VIRGINIA MEDICAL EDITORIAL

## Physicians and the Law Against Drug Abuse

**T**HERE are few issues in our country more pervasive, more destructive, and more heart-rending than the problem of substance abuse.

The problem reaches into every level of American life, from children of elementary school age experimenting with prescription, non-prescription, or illicit substances, to the high school student who regularly supports a heavy drug-use habit, to workers who endanger themselves and others through drugs, to professionals in every area of society.

The medical community realized earlier than most the extent of the drug problem and has been deeply involved in efforts for many years to combat this menace. But in recent years even the highly trained and dedicated medical practitioner has become vulnerable to substance abuse. Fortunately, the number of health care professionals who have used illicit drugs or participated in the illegal drug industry remains exceedingly small, perhaps a reflection of the high standards of the profession and the heightened awareness of the danger of such abuse.

But those high standards, self-imposed as they are, demand something extra of health care professionals. They demand, on the one hand, diligence in helping to prevent and detect substance abuse by fellow professionals, and on the other hand, compassion in helping colleagues deal with the tragedy of substance abuse.

From a law enforcement standpoint, there are several tactics that health care professionals can adopt that will help with the problem. With the help of Virginia law enforcement officers, we have identified several sources of illegal diversion of prescription drugs, and the health care professional can play a key role in preventing that diversion. We have found that many unscrupulous people victimize innocent doctors and pharmacists by concocting false symptoms to obtain drugs for abuse or by forging prescriptions to obtain drugs for abuse and diversion.

Careful internal control of prescription blanks can help substantially in alleviating the problem, and continued sensitivity in prescribing will also be valuable.

Careful inventorying of drugs and tight security for drug supplies will also lessen the danger of drug diversion.

Certainly the record is clear that Virginia medical practitioners care very deeply about this problem and have been among the leaders in our communities in devising treatment and prevention strategies. No one can accurately estimate the number of hours donated by the profession to drug counseling and treatment organizations, to education programs and to community leadership. The value of those contributions of time and talent is literally immeasurable.



The advice and guidance I have received from health care professionals and organizations have been a key to the success of our legislative and law enforcement efforts to combat drug abuse and drug crimes in all their varied forms.

The need is great. Fully half of the 400,000 inmates of our nation's prisons were regular drug users at the time they committed the offense for which they were jailed. One study of 460 male addicts indicates that they committed an average of more than 5,000 crimes a month during their criminal careers, and more than 20% of those surveyed routinely carried guns or other weapons. There can be no doubt of the link between drug abuse and violent crimes.

There can be no better defense than the continued close cooperation between the medical community and the rest of our society toward effective law enforcement, treatment and prevention strategies.

We face a lost generation of drug abusers if we cannot halt the growth of this tragedy. The medical community represents a lifeline to save that generation. I am proud of the opportunity to work closely with The Medical Society of Virginia and other health care groups and promise a continued close working relationship for the future as we strive to eliminate substance use and abuse.

GERALD L. BALILES,  
Attorney General of Virginia

## Mission Accomplished

**T**HE IDEA that I was to have an education and live a life of service to humanity was formed a few years before I was born. My Dad was one of ten children, living on a poor Carroll County, Virginia, farm—working hard, wearing brogan shoes and hand-made clothing. He had a dream of going to college, getting an education and getting into some sort of business. So he found his way to a college in Tennessee. The authorities laughed at him because the only education he had was about the fifth- or sixth-grade level obtained by a few months of attendance during several winters at a one-room grade school. Ridiculed and sent away empty-handed, he made a determined resolution: "If I ever get married and have a son, he is going to get an education." He met and married a country girl who had endured hardships similar to his.

I was born on June 1, 1900. One of the first things I can remember was that I was to get an education. I was quite young when I was taught letters and figures, and when I was 6 years old and allowed to walk two and a half miles to a one-room school, I could count and read a few simple sentences. Dad looked ahead and knew that he would have to get away from home to earn money to send me to school. He worked for the Norfolk & Western Railroad a few years and then went to Oregon, where he found employment on a ranch—made \$40 a month, sometimes slept on a hay wagon. He was still dreaming of being able to help me get an education.

Dad, Mamma, my grade-school teacher, and a friend from nearby Galax who was a college gradu-

ate encouraged me to go on to high school. Some of my schoolmates could not understand that. Why should I leave the quiet life, Sunday school, church and peaceable community to get more education? Why did I need it? A few got the idea that I was just trying to get above them—and they did not like it.

High school was to be difficult for me. It meant walking four and a half miles through mud, bad roads, fields and woods during all sorts of weather. I entered Galax High School in the fall of 1916—carried my books and noon lunch in a long book bag and studied late at night by the dim light of an oil lamp. I was a normal boy and, naturally, learned to like some of the high school girls. It hurt me to see the other boys take their girls out in automobiles for rides. I contacted my Dad a number of times and begged him to get us an auto so that I could be like the other boys. His answer was always firm: "No, I'll help you get an education and let you buy your own car." I made good grades and the four years passed rather quickly. My graduation class was one of five boys and five girls.

Of course I was going to college—but what course was I to follow? Engineering? My grandfathers and my Dad were very mechanical. They could build almost anything they needed in a blacksmith shop. One even built a mill to grind meal with a water wheel. Dad built his house, even baked the bricks for the two chimneys. I inherited the gift of being able to build things—windmills, water wheels, toys, paper gliders, kites, etc. I was also deeply interested in medicine. A few doctors were idols of mine. One had treated me for a bad case of

typhoid fever, and another had cared for me when I was badly injured in a runaway-horse-and-wagon accident. I decided to become a doctor.

I spent part of my vacation at a job in Galax—walked the four and a half miles to help build an “extract plant,” where the sap and essence of chestnut wood and oak bark (tannum) were extracted to get tannic acid for tanning leather goods, and after it was built I worked there, packing the wood and bark. While I was there, a young man told me that he could get me a job milking a cow for one of the professors at the College of William and Mary, and that I could go from here 50 miles to Richmond to study medicine.

Mamma packed my little multicolored, hump-backed trunk with clothing and made me take \$18, down to the last penny of her savings. This made a total of \$118 I had for entering college. I also had a small scholarship to my credit. I boarded a train at Galax at 11 AM on September 13, 1920, and rode six different trains and one streetcar to arrive at Williamsburg on September 14, twenty-four hours after leaving Galax.

I found myself to be a total stranger in the crowd at the railway station. Then a very nice boy came up to me and took me to my room in the Institute Building (better known as “Hell’s Half Acre”). My money disappeared in a flash at the admissions office. I was introduced to the professor’s cow, and all I had left was a determination to get a college education.

My college work was hard, and physical work—milking a cow, digging in the college garden, waiting on tables, cutting wood, pumping out flooded basements, and cleaning the dining room for college dances—required lots of effort. I managed to make enough to pay for my board and a few necessary expenses but many times walked down Duke of Gloucester Street with empty pockets.

Dr. Robins from Stuart Circle Hospital in Richmond visited our pre-medical class and gave us a message I have always remembered. He said, “If you are going to be a doctor just to make money, you are a failure before you start, but if you are going into that profession to help humanity, you will be a success.” I decided to follow his advice of helping humanity.

I finished my two years of pre-med work, made good grades and entered the Medical College of Virginia in the spring of 1922. I roomed at a boarding house, was conservative, studied hard, went to church and lived a normal life along with the other medical students. I spent my vacations at home and looked after the farm while Dad helped

pave the streets of Galax. I passed the final examinations in regular order.

Planning for graduation was a pleasant occasion except for one sad note. Dad and Mamma had worked so hard to see me through but did not feel financially able to come to Richmond to see me get my MD degree. I joined the class at St. Paul’s Church to put on cap and gown for the baccalaureate sermon. What a surprise when a classmate came in and announced to me, “Your father and mother are here! I just took them over to your room. They will be here in a few minutes.”

It was one of the happiest days of my life to walk down the aisle and see the triumphant smiles on the faces of my parents. A family of my good friends in Richmond had secretly contacted my people and arranged to have them as their guests during the commencement. Dad was so proud of my diploma that he carried it, unwrapped, back to Galax.

I did my intern work at Memorial Hospital in Richmond. This was a hard but useful year—doing surgery, going to homes to deliver babies, riding city ambulances at 50 miles an hour, and treating all kinds of diseases. I made good grades on my state board examinations—90% on four of my subjects. It was great to know that I was a licensed doctor.

I bought a Model T Ford coupe, one of the last off the assembly line, and enjoyed a few months of private practice at Palmyra, Virginia, then moved back to Carroll and Grayson Counties and married a country girl who was a college graduate and school teacher. She has stood by me, endured my many idiosyncracies, and has taken care of my medical records down through the years. I worked a year with the Virginia State Health Department and settled in Floyd, Virginia, for my life’s career.

My practice as a small-town and country doctor has been very general and, without bragging, I feel that I have been a success—hard work but one of Christian service to humanity.

A Bible verse, “But seek ye first the kingdom of God, and His righteousness, and all these things shall be added unto you” (Matthew 6:33), is on top of my glass-covered desk. I have tried to follow this advice and have found that it works.

My parents lived to see their dream come true. They saw me a successful physician located in a nice brick home and office. They were proud to know that I lived a life of service to mankind, charged reasonable rates for my services, and treated patients whether they had money to pay or not.

F. CLYDE BEDSAUL, MD

Box 115  
Floyd VA 24091



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# VIRGINIA MEDICAL OBITUARY

## John A. Payne, MD

Dr. John Abb Payne, for almost 50 years a family practitioner in North Carolina and Virginia, died February 11 at the age of 77. He had been in semi-retirement because of ill health since 1980.

Born in North Carolina, Dr. Payne was reared in Virginia at Appamattox and Culpeper. He was graduated from the University of Richmond and the Medical College of Virginia, then trained at St. Vincent's Hospital, Norfolk. In 1935 he established his office in Sunbury, North Carolina, near the Virginia line, and thereafter treated patients in both states.

As a member of the Tri-County Medical Society, he served on its Executive Committee and as an alternate delegate to The Medical Society of Virginia's meetings, and he had been vice president of the North Carolina Medical Society.

## A. M. Edmonds, MD

Dr. Albert M. Edmonds, long-time Richmond pediatrician who retired to Kilmarnock in 1982, died of a heart attack during a deep-sea fishing trip off Virginia Beach on July 31. He was 65 years old.

A native of Lyons Falls, New York, Dr. Edmonds earned both his bachelor's and medical degrees from Syracuse University and trained at Johns Hopkins Hospital and Children's Hospital, New York. He conducted a private practice in Richmond for 17 years, then became director of pediatric education at Richmond Memorial Hospital, where the newborn intensive nursery is named for him.

Dr. Edmonds had been a member of The Medical Society of Virginia and the Richmond Academy of Medicine for 36 years and belonged also to the American Academy of Pediatrics, the Richmond and Virginia Pediatric Societies, the Southern Medical Association, and the American Medical Association.

## Donald M. Levy, MD

Dr. Donald Marvin Levy, 54, died July 9 in a Suffolk Hospital. He had conducted a private practice in neurology in Norfolk since 1962.

Born in Baltimore and reared in Suffolk, Dr. Levy was graduated from Suffolk High School and went on to the University of Virginia and its School of Medicine. He trained at Grace-New Haven Hospital in New Haven, Connecticut, served for two years in the Navy at the US Naval Hospital, Philadelphia, then performed his residency in neurology at Baltimore City Hospitals and Johns Hopkins Hospital.

Dr. Levy was an associate professor of neurology at Eastern Virginia Medical School and had been secretary-treasurer of the Virginia Neurologic Society. He belonged also to the Norfolk Academy of Medicine, The Medical Society of Virginia, the American Academy of Neurology the Tidewater Neurologic Society, and the American Medical Association.

## W. F. Mitchell, MD

Dr. Walton F. Mitchell, retired family physician who was active in Craig County civic affairs, died July 18 at the age of 81.

Born in Bedford County, Virginia in 1902, Dr. Mitchell earned his medical degree at the Medical College of Virginia and moved to Craig County in 1927. There he became a community leader, serving on the county's school board and the New Castle town council. In 1972 he donated ten acres to the county for a recreational and educational camp, and when he retired in 1975 the facility was named Camp Mitchell in his honor. Democratic party politics claimed his active participation, and he was a charter member and former president of the New Castle Lions Club.

Dr. Mitchell's membership in the Roanoke Academy of Medicine and The Medical Society of Virginia spanned 56 years.

# WHO'S WHO

New president of the American Academy of Occupational Medicine is **Dr. Ernest M. Dixon**, McLean, who is head of his own firm, Environmental Health Consultants.

Formerly corporate medical director for the Celanese Corporation, Dr. Dixon earned his medical degree at the University of Virginia and a doctorate in science from the university of Cincinnati. He is a member of the board of directors of the UVa Medical Alumni Association and director of occupational medicine for the Fairfax Hospital.

After 38 years of partnership practice, the **Doctors Pennington, Margaret A. and William A.**, have retired. They cleaned out their joint office in Buckingham Courthouse and headed for the home that drew them to Virginia to set up their practice in 1946.

Margaret Allen was born in Kentucky, William Pennington in Newport News. They met at the Medical College of Virginia and were married after both received their MDs. After residencies for both and World War II service for Dr. William Pennington, they went looking for a likely place in which to establish a country practice. When they got to Buckingham County, Virginia, and saw a fine old house named "Col Alto," their search was ended. Col Alto was built in the mid-1800s by a descendant of Virginia's earliest settlers, Col. Thomas Moseley Bondurant, who made his mark by accumulating land, exporting tobacco and serving in the state legislature.

Dr. William Pennington caught the legislative fever himself. In

1960 Buckingham County voters elected him to Virginia's House of Delegates, and they returned him to that office three times. Nor has Dr. Margaret Pennington been content to make her mark in medicine alone. An accomplished artist, she has recorded in pen and ink historical structures and sites all over Buckingham County. Two hundred of these drawings were published in a book titled *The Courthouse Burned*—. With the artist's permission, many of these drawings have appeared in VIRGINIA MEDICAL.

To **Dr. Julia Terzis**, director of the Microsurgical Research Center at Eastern Virginia Medical School, went the honor of hosting the 30th Annual Plastic Surgery Council Meeting held recently in Norfolk. Her selection was made by the Council, which is comprised of plastic surgeons who are chiefs or department chairmen or have contributed significantly to research.

In what the Culpeper *Star-Exponent* reported as "a difficult decision between three enthusiastic nominees," a selection committee named **Dr. Morton P. Chiles III** to fill a vacant seat on the Culpeper County school board. Dr. Chiles is a family physician.

When six Radford, Virginia, citizens were honored recently by the local Rotary Club, one of the honorees was **Dr. Theron H. Hass**, obstetrician-gynecologist who has been president of Radford Community Hospital and was Radford's vice mayor in the early '60's. He and the others were named Paul

Harris Fellows of the Rotary Foundation of Rotary International.

Virginia's high school athletic directors tapped **Dr. H. Joseph Williams** of Staunton for their annual distinguished service award. Since 1948 Dr. Williams has provided for the medical needs of athletes in the Staunton-Augusta County area.

"Don't retire unless you have to," advises **Dr. John G. Ringler** of Manassas. "I miss it. I miss the people and all the kindness they showed me throughout the years. I wouldn't have retired if I hadn't had to because of my health."

He was talking to newspaper reporter Stephanie Walker, whose feature story on Dr. Ringler appeared in a recent issue of the *Manassas Journal Messenger*. Poor health forced Dr. Ringler's retirement after 35 years of serving the Manassas community as a family physician.

A native of upstate New York, Dr. Ringler was graduated from Notre Dame University and the Georgetown University School of Medicine and chose Manassas as his place of practice. "I couldn't have picked a better place," he told his interviewer.

In 1954 he founded the Manassas Medical Center. It was the only medical facility in Prince William County and the only real emergency room between Arlington and Charlottesville. He and the physician who joined him in partnership, **Dr. Robert A. W. Latimer, Jr.**, were on call 24 hours a day seven days a week. The Center prospered, drew specialists to the area, and now it serves a greatly expand-



ed population. "We both settled here to be country doctors and became city doctors without even moving," observed Dr. Latimer.

Dr. Ringler married Lelia Dow, daughter of a Manassas pharmacist, and they have two daughters, Helen, a senior at Notre Dame, and Mary Lea, now a wife and mother in Massachusetts.

**Dr. Jesse J. Bates**, who has conducted a general practice in South Boston for 35 years, was honored by the Mary M. Bethune Alumni Association with its medallion for "outstanding work with the group and in the community."

The Arlington County Medical Society tapped one of its past presidents for the 1984 Welburn Award. He is **William D. Dolan**, pathologist.

Elected to membership by the American Orthopedics Association was **Dr. Frank C. McCue III**, professor of orthopedics at the University of Virginia and a specialist in sports medicine and surgery of the hand. Dr. McCue was one of 18 physicians invited to join the association, which was founded 97 years ago and has 525 members in the United States and Canada.

New chief of staff at Montgomery County Hospital is **Dr. Alex A. Tan**, Blacksburg surgeon.

For his outstanding work in the field of diabetes, **Dr. H. St. George Tucker**, Richmond, was given the Upjohn Award by the American Diabetes Association at its recent meeting in Las Vegas. The award is

given yearly to a top medical educator in diabetes. Professor of medicine at the Medical College of Virginia, Dr. Tucker also recently received the college's faculty achievement award. He has been at MCV since the 1940s.

Appomattox celebrated **Claude G. O'Brien** Day on a fine day in June, and a plaque was presented to Dr. O'Brien by the mayor for his "outstanding service during the past 50 years in the practice of medicine." Dr. O'Brien was reared in Appomattox and is a graduate of the University of Richmond and the Medical College of Virginia. Except for a two-year stint in the Navy, he has practiced medicine in Appomattox since he completed his GP training in 1934.

**Dr. Harold William Markham**, Culpeper, has been elected to the board of directors of Blue Cross and Blue Shield of Virginia.

**Dr. George E. Chappell**, Halifax, retired early this summer and promptly took off on a cross-country camping tour that wound up in Alaska. His wife was with him, they were in their mobile home, and Alaska is the home of a son, Ronnie, and his family. Writing in the *South Boston News and Record*, reporter Sylvia O. McLaughlin described Dr. Chappell as "excited about canoeing Alaskan streams with his son, who is also an ardent outdoorsman."

After coming to medicine a bit later than most, "Chip" Chappell gave 30 years of doctoring to the Halifax community. He studied for a few years at Randolph Macon

College, then enlisted in the Navy for World War II, winding up as a flight instructor in New Orleans, where he met his wife, Ruth. After discharge he was a partner in a restaurant in Richmond for a few years, then the yen to be a doctor struck him. He returned to Randolph Macon for his diploma, moved on to the Medical College of Virginia to take his MD, and set up practice in Halifax, where he's been ever since.

**Dr. Kenneth E. Heatwole**, Charlottesville, was the 1984 recipient of the American Lung Association of Virginia's highest volunteer recognition, the Douglas Southall Freeman Award. Dr. Heatwole is assistant director of the tuberculosis service at the University of Virginia's Blue Ridge Hospital.

**Dr. Juan Montero**, Chesapeake, has been appointed by the American Medical Association's board of trustees to a two-year ad hoc Committee on Foreign Medical Graduates, which will study the problems of FMGs in this country. Dr. Montero himself was graduated from the College of Medicine, Cebu Institute of Technology, Cebu City, the Philippines.

Three Medical Society of Virginia members took office at the annual session of the Seaboard Medical Association. They are **Dr. Willette L. LeHew**, president, and **Dr. William V. Tynes II** and **Dr. George L. B. Grinnan**, vice presidents. All practice in Norfolk. Dr. Tynes has another new office—president of the medical staff of Medical Center Hospitals in Norfolk.

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## From the President: Who ever said life's fair?

**A**NYONE who has read the newspapers in the past year knows that there is a tremendous controversy brewing in this country over organ transplantation.

Initially, everyone was amazed at the success of skin, bone and kidney transplants. With the first two, there was not much concern or controversy, as many people could, and did, supply these needs for themselves. In case of kidneys, frequently a family member supplied one. When HLA typing became more common, other donors were available, but never enough to meet the transplant need.

As technology continued to advance, we arrived at the point where experimentation with heart, liver and lung transplantation was succeeding. Obviously, live donors were out of the question. So, some entrepreneurs suggested getting organs from overseas—at a price.

The medical profession and the government became alarmed at this turn of events. Life is cheap in some Third World countries, and there were serious concerns about the possible ramifications of purchasing human organs. Legislation has been introduced to mandate government control of the supply and also to outlaw the sale of organs altogether.

We all remember the problems that arose when renal dialysis became more accessible in the 1960s. Citizen committees were set up to determine who received dialysis and who didn't. They were accused of discrimination. To solve the problem, national legislation was passed which encompassed both kidney transplants and dialysis. When the program was initiated a little over a decade ago, the projected cost was \$200 million annually with



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11,000 applying for dialysis and transplant. Today the program costs \$2 billion for 65,000 patients.

Before this, approximately 75% of the dialyzed citizens continued to work. Now it's estimated that only 25% continue to work; the others live for dialysis only. Is the medical profession to blame for this large expenditure of money? Should they stop developing new techniques or making advances in diagnosis and treatment if they are to be criticized because of the costs?

Now we are faced with the same dilemma with heart, liver and other potentially transplantable organs. Where are the organs coming from, and who is going to decide who gets what? Who will pay the enormous cost? Will the government decide these issues and control your organs when you die? Will they give the organs to people who are 75 years old, or someone 21 years old? Does a mental defective who is 15 years old but otherwise healthy receive a transplant before an older person? Maybe there should be a lottery! Obviously, bias will enter into any kind of decision made by whatever group chosen to make it, whether private or governmental. Personally, my confidence and faith in the government making better decisions than private citizens is low. It has intruded in our lives in too many ways already.

Now is the time for physicians to be thinking of these issues and working toward responsible solutions. We must get involved. If not, one day we will wake up and Big Brother will have made that decision.

**C. BARRIE COOK, MD, President,  
The Medical Society of Virginia**

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# LETTERS

## **Finds clonidine effective in treating narcotic addicts**

Peninsula Drug Rehabilitation Services, Inc. (PDRS, Inc.), a drug abuse program located in Hampton, Virginia, began to use clonidine in the treatment of narcotic addiction after reviewing research successes on narcotic addicted patients. This report is based on our experience using clonidine. I feel that the information may be useful to others who are looking for alternative treatment modalities for narcotic addicted patients.

Dolophine, a synthetic narcotic, was developed by German chemists during World War II as a substitute for morphine as their supply of opium was restricted. (Dolophine was reportedly named for Adolph Hitler.) Methadone was developed from dolophine in this country so that it could be administered by mouth. In 1965 Nyswander and Dole used methadone in detoxification of heroin addicts with some success.<sup>1</sup> Later the federal government and various state governments established methadone centers to treat confirmed narcotic addicts. This was to be done in two ways: by detoxification, using diminishing doses of methadone over a period not to exceed 21 days or by daily maintenance doses to keep the addicts away from street drugs and out of crime.

Methadone relieves many of the symptoms of narcotic withdrawal and the long-lasting effect enables the patient to maintain a stable lifestyle. However, methadone is just as addicting as other narcotics, and it has been our experience that detoxification from methadone is just as difficult for the patient as withdrawal from other narcotics; because of this difficulty, we have had limited success with methadone detoxification.

In 1978, Gold and coworkers<sup>2</sup> at Yale found that the antihypertensive drug clonidine (catapres), when given to narcotic addicts, in some cases took away their desire for narcotics and relieved the symptoms of withdrawal. The mode of action of clonidine in alleviating the desire for narcotics and relief of withdrawal is thought to be an antiadrenergic effect in the cuneate area of the brain<sup>3</sup>. Many reports are in the literature of treating drug addiction with clonidine in the hospital.

Two years ago we decided to try clonidine on an outpatient basis in certain selected cases. The client had to be on a low dose of methadone, 20 mg or less; blood pressure had to be within a normal range sitting, standing and lying down; and, most important, the client had to be motivated to get out of the drug culture and become drug-free. The dosage of clonidine was low (0.05 mg). After a test dose, the client was monitored for symptoms of hypotension. If there was no adverse reaction, the client was given 0.05 mg three times a day at about eight-hour intervals, checked daily in the clinic, and the dosage was adjusted as indicated. This was continued over a period not to exceed ten days.

Drug rehabilitation counselors at PDRS, Inc., were surveyed as to the current status of the clients who completed either methadone or clonidine detoxification during a ten-month period. Based on counselors' knowledge, the following information was gathered.

Forty-four confirmed addicts were admitted to voluntary methadone detoxification. At the time of this writing, we could confirm that four are drug-free, seven returned and were placed on maintenance, three were in jail, 11 were using street drugs, two died, and 23 were lost to followup and could not be contacted at their address, by telephone, or by mail. This is a 9% success rate.

During the same ten-month period, 11 clients completed clonidine detoxification treatment. In one case, the clonidine was not tolerated because of hypotension. In the other ten cases, three are documented drug-free and the other seven returned to methadone maintenance. This is a 33 $\frac{1}{3}$ % success rate compared to the 9% success rate with methadone detoxification during the same period.

The methadone dosage is given once a day mixed in juice, and must be taken in view of the nurse. The clonidine dosage (tablet) is taken at home three times a day; blood pressure is checked once a day by the nurse before the tablets are given. It may be that more attention is paid to the patient on clonidine and possibly is a factor in the relative success of this medication, although the nurses usually inquire of all the patients how they feel.

The drug manufacturer has not recommended the use of clonidine in the treatment of narcotic addiction.

tion, nor has the Food and Drug Administration approved its use for this purpose. Clonidine is certainly not a cure for narcotic addiction, but we feel that it has a higher potential for success for motivated patients than a methadone detoxification. In dealing with drug addiction, success is measured in very small quantities. We shall feel more comfortable using clonidine to treat narcotic addiction on an outpatient basis when the manufacturer recommends its use for this purpose and the Food and Drug Administration approves it.

**Barnes Gillespie, MD**

Medical Director,  
Peninsula Drug Rehabilitation Services, Inc.  
804 West Mercury Boulevard  
Hampton VA 23666

1. Dole VP, Nyswander ME: A medical treatment for diacetyl-morphine (heroin) addiction. JAMA 1965;193:646
2. Gold MS, Pottash AC, Sweeney DR, Kleber HD: Opiate withdrawal using clonidine: A safe effective and rapid non-opiate treatment. JAMA 1980;243:343-346
3. Bourret JA: Effects support noradrenergic hyperactivity hypothesis. Hospital Formulary, April 1981, p 431

### **New eating disorders program opens at University of Virginia**

The July issue of VIRGINIA MEDICAL with the group of articles on eating disorders was of particular interest here at the University of Virginia because we have a new Eating Disorders Program for the treatment of patients with anorexia nervosa or bulimia. Begun in November 1983, the program consists of an inpatient/outpatient clinic, with hospitalization available on a unit of 20 beds. The staff consists of psychiatrists, psychologists, nurses and nutritionists under the supervision of Dr. John Buckman, medical director. The treatment team has developed protocols for both disorders, and they are working well with our patients.

We welcome the inquiries of Virginia physicians who have patients with anorexia or bulimia.

**Judith K. Sights, PhD**

Consultant, Eating Disorders Program  
Department of Behavioral Medicine and Psychiatry  
Blue Ridge Hospital  
Charlottesville VA 22908

### **Dr. Corcoran's retirement stirs memories**

In the Who's Who section of VIRGINIA MEDICAL's June issue was a report that Dr. David B. Corcoran of Suffolk had retired.

At the old Long Island College of Medicine on Henry Street in Brooklyn in the 1939-1940 years, I was at 30 years of age a freshman and David, I am sure much younger, was a senior. In late April of this year in Williamsburg, during a three-day course in electrography, the man sitting next to me was a Dr. Baylor, who was in David's class.

**(The Rev.) N. H. Wooding, MD**

11 South Main Street  
Halifax VA 24558

### **Suggests quality assurance section for Va Med**

I would like to suggest that VIRGINIA MEDICAL include a section for quality assurance. This could be a voluntary effort by physicians and hospitals to demonstrate their continued support for maintaining satisfactory medical care standards without a mandate from the federal government. These reports could be provided by hospitals and physicians as well as specialty societies, such as the Cardiac Society for Angiography, which was organized several years ago to provide a registry for hospitals to report major complications secondary to cardiac catheterization. This allowed each institution to compare results with the national standard and provide a method of improving quality of care.

It is unfortunate, in my opinion, that so much has been spent on PSROs, with the major focus being on hospital length of stay rather than the politically sensitive issue of quality of care. In any event, a voluntary section in VIRGINIA MEDICAL devoted to brief reports from individuals and hospitals or specialty societies would be a step forward in reestablishing the credibility of the medical profession rather than continued interference from the federal government.

**Charles L. Baird, Jr., MD**

205 North Hamilton Street  
Richmond VA 23221



The Department of Neurology  
at the  
Medical College of Virginia,  
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announces  
the retirement of  
Dr. Cary G. Suter as Chairman  
and the appointment of  
Dr. John B. Selhorst as Acting Chairman.

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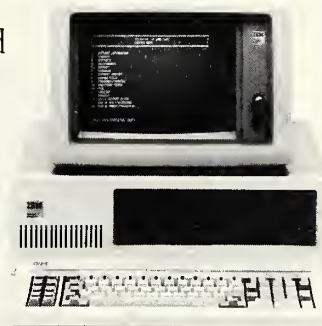
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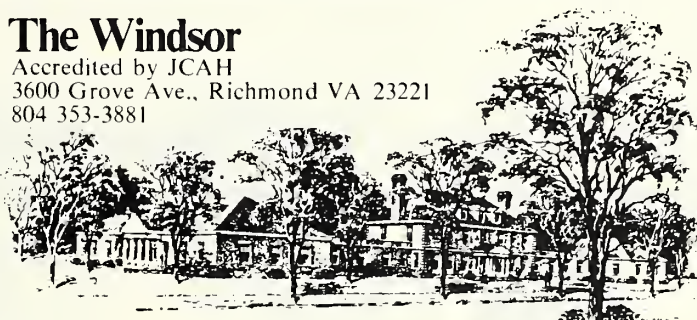
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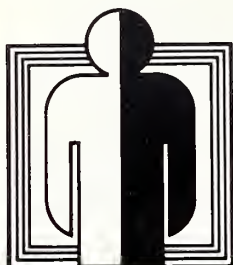
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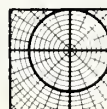


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# THE RISK-BENEFIT RATIO IN MEDICAL HERO-WORSHIP

John A. Owen, Jr., MD  
Charlottesville, Virginia

**T**HE Bible says, "A prophet is not without honor, save in his own country and among his own people". If this be true, what greater honor can befall a professor than for his own students, at his own medical school, to come to him and say "Give us a lecture"? I count this the highest honor of my 24 years on this faculty and I thank you most humbly for it.

We are gathered here to celebrate the memory of a great and gracious lady, one who was beloved by more than 20 successive classes of medical students. Unfortunately, the Class of 1984 never knew her, and it is only fair to ask "Who was Annie Lipscomb and why do we admire her?" Let my answer to this question serve to introduce my topic.

On the wall of the medical library a bronze plaque reads, "The Annie G. Lipscomb Memorial Lecture. In honor and recognition of the devoted service and

This paper is based on the Annie G. Lipscomb Memorial Lecture, delivered by Dr. Owen on February 9, 1984, at the University of Virginia. Address correspondence to him at the Department of Medicine, Box 242, University of Virginia School of Medicine, Charlottesville VA 22908.

warm friendship given so unselfishly to medical students by Annie G. Lipscomb, Secretary to the School of Medicine. Established by the Class of 1968."

What is the story behind this? A member of the Class of 1968 told me once: "To us, Miss Annie was the Medical School. She seemed to know everything about each one of us, she clearly loved us all, and she had complete confidence in our future success."

Those are fine words, and we could leave it at that, but sometimes a picture is worth more even than fine words, so accompanying this text you will see a portrait of Annie Lipscomb at the height of her powers, sketched by Dr. James N. Pope, a member of the Class of 1966 and now a surgeon in Lynchburg.

At first glance this appears to be a caricature of Dr. Kenneth

Crispell, then Dean of the Medical School, in all his imperial regalia. However, this striking likeness of Miss Annie depicts her as the real power behind the throne, cueing the Dean effectively in everything he does. Note the significance of the book she holds in her left hand, that unique *vade mecum*, "How to Run the Medical School".

It is quite clear, then, that generations of medical students have looked upon Miss Annie with something close to idolatry. She embodied all that was admirable about the institution, she had magical and beneficent powers far beyond those of any ordinary secretary, and those who believed in her survived. It was truly a form of primitive worship, indeed perhaps the finest example of medical hero-worship that one might ever find for the purposes of this discussion. But there are many such examples. Indeed, with a little imagination one can see each succeeding generation of medical students instinctively focusing on someone to venerate, and (with the passage of time) automatically earning itself the veneration of the next generation.

But why "automatically?" Why does this universal drive toward hero-worship pervade the entire medical profession? An answer came to me four years ago when there returned for its tenth reunion



the Intern Class of 1970-71. Now everyone knows the intern year to be the most stressful, the most gruelling, the most totally depressing experience that a human being can undergo and still live. Why would anyone want to remember the tenth anniversary of that year, and to travel many miles back to Charlottesville to celebrate it?

As I pondered this enigma I postulated that probably the only thing that had kept these former interns going through that crucial year was the overwhelming need to believe—to trust that somewhere in this fantastic merry-go-round of non-stop admissions, multiplying problem lists, terminal disease and ubiquitous death, there existed wise physicians who had seen it all, who knew what to do, and would help them see it through. And ten years later, being now expected to give that same measure of wisdom to others, they perhaps felt, like Antaeus, a need to touch the source of their strength, to return to the spot where the saving grace of hero-worship had helped them through a rough time.

My hypothesis, therefore, is that medical hero-worship is a form of behavior which has great survival value for individuals going through stressful experiences. It must be extremely effective or else why should it be so prevalent? But like any therapeutic modality, it has risks as well as benefits, and we should attempt a balanced evaluation of both.

**T**O BEGIN with the most obvious benefit, students learn better if they hero-worship their instructors. It may be more a rationalization than rational to believe that great wisdom dwells always in great men. But since medicine is still an art as well as a craft, surely we learn not only by doing what we see our superiors do, but also by being what we see them to be. In the frenzy of Saturday night in the emergency room, in the high drama of the operating room, perhaps most of all during the long midnight hours up on the ward, the student learns not only the skills of medicine but the grace



under pressure that is best absorbed from those whom one deeply admires. When the Code 12 battle has been fought and lost in a room down the hall, when resident and student walk wearily back to the office and sit down and look at each other, and when the resident slowly picks up the phone and dials a number, and with infinite compassion says "Mrs. Jones? I am afraid we have some bad news for you," then it is, if never before, that the student really begins to introject into his nature some of the virtues of his preceptors. He must. How else can he survive?

Medical hero-worship is akin to military hero-worship but with a difference: While all privates don't realistically expect to become generals, all medical students sooner or later expect to become physicians. And that means that they too will be making those terrible midnight calls, year in and year out for the rest of their lives. Instinctively they reach out, while they can, to incorporate all those healing strengths that they see in others and that they desperately need to see in themselves. In summary, to survive in medical school, students must hero-worship their teachers.

The second benefit comes a little later in one's professional career. Physicians soon find that they

are better healers if they look upon their patients with appreciation and respect, if not actual hero-worship. (Yes, I know, the traditional wisdom is that patients look upon their doctors with hero-worship, but the converse is also true.) Probably no other profession gives its practitioner so many unsought opportunities to see what the naked human spirit is made of, under all sorts of terrible adversity. And what we see is not just cooperation and courtesy and compliance, those textbook graces, but searing instances of sublime devotion and self-sacrifice and a stoic courage in the face of impending doom.

Over and over, our professional lives are illuminated by the nobility of ordinary people. When we have sat down at a patient's bedside and said those terrible little words: "Your biopsy shows malignant cells," we cannot avoid seeing that sudden constriction of the countenance, that intense searching look, that inward tensing up of the spirit; we know that inside the patient the clock stops and from that moment on things will never be the same—and we did it. And when the discussion is over we always walk away thinking, "There but for the grace of God go I." For we have looked into the soul of an ordinary person and glimpsed the angel inside. And from that unforgettable encounter we take increased devotion to our life's work. Truly, to survive as physicians we must hero-worship our patients.

A final benefit, reserved for those in the teaching profession, is that we teach better if we look upon

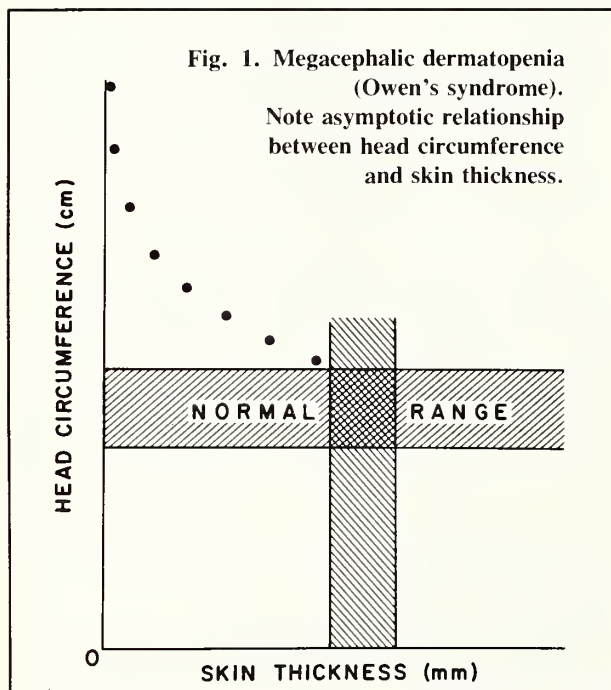
our students with admiration, if not a certain hero-worship. That, I am sure, must sound like total heresy, except for those who attend the faculty meetings each September when the vital statistics of the entering class are proudly recounted. When we peruse those astronomical GPAs and MCATs we invariably turn to each other, shaking our heads with stunned delight, and invariably, we whisper, "I could never have gotten into medical school if I had to do it again today." And so from year to year we look forward with eager anticipation to the bright new men and women who will keep us learning even as we help them learn. The legacy Miss Annie left, therefore, is precious also to us of the faculty: To survive as teachers we must believe in our students.

In summary, therefore, what is hero-worship after all but an expectation of finding the best in others? It is a willingness to see the heroic in everyone, even in the most contemptible, an a priori faith that all will go well because the human spirit will rise to any occasion and surmount all obstacles to heroic performance.

**B**UT IF medical hero-worship has such tremendous benefits, how could there possibly be any risks? And if there are, can we find any way to avoid them?

One quite obvious risk is the dark side of perfectionism, the belief that anything short of the truly heroic is failure. The legendary hero is always in the limelight, always on center stage, and always triumphant. Ergo, he is far superior to ordinary humanity and, ergo, the medical profession rejects vigorously any notion that it might be made up of ordinary human beings. Perhaps that is why as many as 10% of physicians, at some time during their professional lives, become impaired by addiction to alcohol or to other drugs. Characteristically, the first drug ingestion is an ill-advised effort by the physician to maintain his indefatigable and omnipotent image when actually not feeling up to par. Once the habit is begun it is sustained by what has been called the M. Deity syndrome, the delusion that drug-taking is not really a weakness but an ingenious utilization of chemical assistance, that the physician can of course stop any time he wants to, and that he can (and will, of course) do it all by himself. When heroism becomes a compulsion the results can often be tragic: Quos deus vult perdere prius dementat.

This professional personality problem actually amounts to a medical syndrome, which is illustrated in Figure 1. I have called it megacephalic dermatopenia, or Owen's Syndrome. I hasten to add that it





is not named after me, but after my wife, who should be credited with the original description.<sup>1</sup> She made the interesting observation that as the head gets bigger the skin gets thinner, and we see this illustrated in the figure. All of the points on this chart were derived from measurements of my colleagues; even my own measurements somehow turned out to fall along the curve, so we still have no "normal controls." Obviously, more work is needed to verify this initial pilot study.

Well, how do we avoid this risk? It can be done, if one is selective as to whom one worships and why. One of my medical heroes is Lewis Thomas, who writes so beautifully and well but perhaps never better than in his introduction to *Cecil's Medicine*:

*I remember a short story from real life which illustrates an aspect of the responsibility of doctoring which does not find an emphasis in many textbooks of medicine. Some years back I was invited to give a lecture on antibiotics at the annual meeting of a county medical society in a remote part of Mississippi. The audience was almost entirely made up of general practitioners and country doctors. For the president of this society, a man in his 40s, this meeting was the major event of the year and one of the major occasions in his professional life; he was being inducted formally into the office of the president and has his speech prepared and ready. Just as the meeting began he was handed a note and left the auditorium to take a telephone call. That was the last I saw of him until three hours later when he came back looking tired and worn out. I knew that he was deeply disappointed to have missed what should have been his own professional triumph and I asked him what had happened. It was a call from the family of an elderly patient of his who had just died, he said. He felt that he ought to be there to help the family and to be useful. He simply had to be there, he said.*

*This was almost 30 years ago, but I have never been able to forget that doctor and his example of good doctoring that evening. It's not quite the same thing as open heart surgery or curing meningitis, but if I were to look for a role model for today's medical students to look at closely I would pick that country doctor in the backwoods countryside of Mississippi, if I could find him.*<sup>2</sup>

But this risk, even if minimized, still has the power to engender an even greater risk: the sometimes excessive price we pay for our heroic efforts to save life against all odds. We see ourselves as the protagonist in the ultimate contest, the Doctor versus Death. It is a contest we cannot always win but also one that we can never lose by default or by giving less than 100%. So we fight on, down to the last breath, to the last flicker of the EKG, to the last EEG spike, with the desperate intensity of a basketball team going down to the buzzer. We give so

much in every contest that when we lose there is nothing left for us. We are worn out, burned out and exhausted, and we never seem to have time or a place to recharge our batteries because the next contest is already beginning on center stage.

We need to be reminded that death means different things at different times, even to the same people. I once wrote an article recommending that when patients die their doctors should take time off to attend the funeral service. I then tried to describe how a simple country funeral might appear through the eyes of a young house officer, concluding:

*Now we will remember him, not just by his pathology but by the impact his life has made upon this quiet corner, these few country miles concentrated on the church and the hilltop. It is a leave-taking, not just for the doctor but vicariously for all the nurses who teased and cajoled him, the orderlies who cleaned him up, the house officers who drew the blood and inserted the tubes, all of whom carry not just a burden of professional disappointment but a personal grief. They miss him, too. For Jones was not just a sick patient, but "basically a helluva nice guy". . .*

*We physicians periodically need to be reminded that our view of death—all purulence, incontinence and flat-line tracings—is not the only view. There is the view from the grove on the hilltop, and a glimpse of that can be a powerful solace to a dying patient and to his family, particularly when they can be reminded of it even in the midst of the purulence.*

*And there is, of course, the personal thanatopsis, for whatever that's worth. Somewhere down the road, a grove on the hilltop waits for each of us. Reflecting occasionally on that destination somehow gives us grace to get through each day, one day at a time.*<sup>3</sup>

THE FINAL risk is hard to put into words. It smacks a bit of evangelism, which is just another way of saying that it's none of my business. But in the world of the spirit, there are some prices that we have to pay for our beliefs. When we devote our entire professional lives to efforts to preserve life, particularly when we work so hard at the very end of life, our actions declare a belief that there is nothing left after life is over. Hero-worship has become almost a religion, eliminating the need for any other kind of religion. But to each of us there comes a time when hero-worship is not enough.

The traditional theology of a physician begins in medical school when he looks up at his anatomy instructor and says, "Gee, Dr. Jones, I've dissected my cadaver from sagittal sinus to plantaris tendon, and I didn't find a soul anywhere." Next the cynical resident, cigarette drooping from the corner of his mouth, who says, "When my patient died yesterday, his kidneys were transplanted into two people

on the Renal Service, his corneas went into the Eye Bank, and his pituitary was harvested for its growth hormone content. The rest of his body is in 100 different slides, blocks, crocks and bottles in Pathology. When the last trumpet sounds they're going to have one hell of a time trying to put that sucker back together again." The final scene shows the cracker-barrel philosophers around the country store discussing Old Doc, and they agree Old Doc doesn't have much truck with preachers, "In fact, I ain't never seen him go to church. But when Judgment Day comes, I'll take my chances on Old Doc getting through them Pearly Gates." Surely we all treasure these charming scenarios, but the fact is that there is nothing in any Christian (at least) theology that says the good doctor will go to Heaven simply because he is a good doctor.

The average physician, of course, doesn't think about theology when he is working night and day to save the life of a dying patient. He does what everyone expects him to do, using every skill and trying every trick in the book in order to sustain life. Even though we all know it's a case of arranging the deck chairs on the Titanic, he arranges them to rectilinear perfection, with the utmost efficiency and optimism. Since this is what is expected of him, clearly this is what he must do. It goes with the territory.

But the intensity with which the doctor combats the death that threatens others is of little avail and may even be counter-productive when the time comes for him to face his own death. As Becker puts it, "We admire the courage to face death; we give such valor our highest and most constant adoration; and it moves us deeply in our hearts because we have doubts about how brave we ourselves should be."<sup>4</sup>

It has been well said that physicians, as a profession, fear death more than all other men. Why? We seem to have forgotten the wisdom of Socrates: On learning that he was condemned to death for his teachings, he commented, "For this fear of death is indeed the pretence of wisdom, and not real wisdom, being the pretence of knowing the unknown; since no one knows whether death, which they in their fear apprehend to be the greatest evil, may not be the greatest good."<sup>5</sup> Are we so much wiser today?

Some outstanding physicians have indeed seemed more fearful than wise when the hour of their own death drew near. One prominent professor at this university, a witty and skillful clinician, suffered a myocardial infarction and at once insisted on seeing his own EKG tracing. When it was finally shown to him, he exclaimed that the arrhythmia

was irreversible and inevitably fatal, turned his face to the wall, pulled the covers over his head, and would not permit anyone to do anything else for him.

But since we still have freedom to choose which heroes to worship, I prefer to worship a man who was with us at the University of Virginia until recently, Julian Beckwith, a great cardiologist, a great teacher, and a great man. Years ago he had irradiation for a tiny melanoma of the retina, and we all thought that would be curative. I saw Julian occasionally for his mild diabetes, and when it worsened I recommended an oral hypoglycemic agent. A few weeks later he developed jaundice; we assumed it was a drug reaction, and I discontinued the drug with much apology. But the jaundice deepened, the ultrasound showed masses in the liver, and a needle biopsy showed malignant melanoma, more virulent than ever.

With a heart full of grief, I forced myself to enter his hospital room, and in a choked voice began to say, "Julian, I am so sorry, I wish to God it had been a drug reaction."

The irrepressible Beckwith grin spread over his face, and he summed up the whole situation in an unforgettable comment, "Don't be sorry for me, John, I've lived a full life, and I've had a lot of fun. The only thing I was afraid of was that I would drag on and on, and get old and sick and senile, and now I know that's not going to happen. I know what I have, and I know how long I've got, and I know that my dying will not be too unpleasant." And then, with that typical Beckwith flourish, he added, "And that's a hell of a lot more than you know, boy!"

It was a hell of a lot more than I knew then, and a hell of a lot more than I know now, or that any of us in this audience knows. But this was the tone he set for the last month of his life, a month in which he stayed at home, welcomed all of his family and friends to visit and regaled them with his anecdotes and reminiscences, his jokes and laughter, his incredible, irrepressible cheeriness. He never really suffered until the last night, and then, thanks to God's own medicine, morphine, it was soon over. We who loved him remember him as Mark Antony said of Brutus, "This was the noblest Roman of them all . . . His life was gentle; and the elements so mix'd in him that Nature might stand up and say to all the world, This was a man!"<sup>6</sup>

**S**O FAR I have honestly tried to inform you of the risks and benefits of medical hero-worship, but alas, I suspect that like so many of my lectures it has all gone in one ear and out the other. Not this



time because of inattention or total boredom, but because, willy-nilly, you are already committed to the medical way of life, and (automatically) to medical hero-worship, and it's too late to turn back. What you feel is what I have felt for so long, the exhilaration so well expressed by Hans Zinsser in his autobiography.

*I remember one dark rainy day when we buried a Russian doctor. A ragged band of Serbian reservists stood in the mud and played the Russian and Serbian anthems out of tune. The horses on the truck slipped as it was being loaded, and the coffin fell off. When the chanting procession finally disappeared over the hill, I was glad that the rain on my face obscured the tears I could not hold back. I felt in my heart then that I never could or would be an observer, and that whatever fate had in store for me, I would always wish to be in the ranks, however humbly or obscurely; and it came upon me suddenly that I was profoundly happy in my profession, in which I would never aspire to administrative power or prominence so long as I could remain close, heart and hands, to the problems of disease."*<sup>7</sup>

If there really is a hereafter, and if Miss Annie Lipscomb's soul lives on there and has a word for you today, it is surely much shorter and sweeter

than all I have been groping to express. With her indomitable grin, she would say simply this:

1. Believe in yourselves.
2. Believe in your patients.
3. Believe in the human race.

This message I therefore pass on to you, under the same conditions by which I received it, that throughout your professional lives you in turn must exemplify it to those who come after you, who will in time, however illogically, worship you as medical heroes, too.

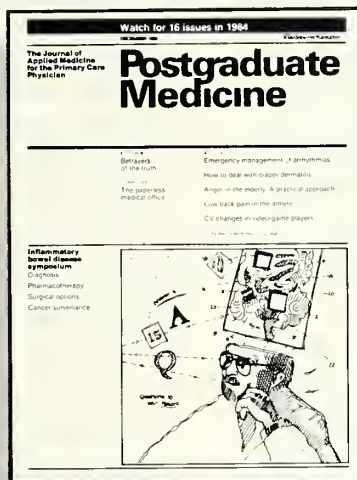
Class dismissed.

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# T U R N

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# Postgraduate Medicine

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# Diabetes Today

William L. Clarke, MD, and Stephen L. Pohl, MD,  
*Charlottesville, Virginia*

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New insights into the roles of genetics, autoimmunity, and environment in the development of diabetes are discussed in this update, together with new ways of delivering insulin and the use of "second generation" hypoglycemic agents. The authors advocate self-monitoring of blood glucose by the patient and frequent determinations of glycosylated hemoglobin by the physician. The only way to reduce morbidity and mortality in these patients, the authors stress, is to keep blood glucose concentrations as near normal as possible.

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SIX years ago an article appeared in *VIRGINIA MEDICAL* describing the state of the art of diabetes care.<sup>1</sup> Since that time there have been significant advances in our knowledge of the pathophysiology of diabetes and marked changes in the techniques of diabetes management. More is known about the etiology of both Type I and Type II diabetes, new dietary recommendations have been suggested, new oral agents are appearing on the market, purified insulin and human insulin are

now available and insulin is now being administered in different ways. Patients are readily accepting self-blood glucose monitoring and physicians are using glycosylated hemoglobin concentrations (HbA1C) to monitor their patients' glucose control. The psychosocial impact of diabetes as a "lifestyle" disease is becoming more apparent, and the education of patients and health care providers is becoming a full-time endeavor.

This up-date highlights some of the important changes that have occurred over the past six years and provides references for an in-depth exploration of individual topics.

At the outset, it is important to restate the recommendations of the American Diabetes Association (ADA) that "the goals of appropriate therapy should include a serious effort to achieve levels of blood glucose as close to those in the non-diabetic state as feasible."<sup>2</sup> Presently, techniques are available to assist in attaining this goal and many diabetic patients are now approaching normoglycemia. Surprisingly, controversy still exists

From the Division of Endocrinology and Diabetes, Department of Pediatrics; the Division of Endocrinology and Metabolism, Department of Internal Medicine; and the Diabetes Research and Training Center, University of Virginia School of Medicine. Address correspondence to Dr. Clarke at Box 386, University of Virginia Medical Center, Charlottesville VA 22908.

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concerning the relationship between glucose levels and the development of micro- and macro-vascular complications.<sup>3-5</sup> The ADA viewpoint, however, now places the "burden of proof upon those who maintain that diabetes control is without effect."<sup>2</sup>

### Classification and Diagnosis

It is now clear that hyperglycemia is a metabolic abnormality common to many syndromes labelled "diabetes mellitus."<sup>6</sup> The etiologies of these disorders, their clinical presentations and their medical management are heterogeneous. In an attempt to clarify the classification of hyperglycemic syndromes, a group of international scientists convened to standardize diagnostic criteria.<sup>7</sup> A summary of their recommendations is shown in Table 1. The present recommendation for the oral glucose tolerance test (OGTT) includes 1.75 gm/kg (ideal body weight) oral glucose with a maximum of 75 gm. Type I diabetes, the term presently used to describe insulin-dependent diabetes (juvenile onset; brittle; ketosis prone), usually presents with unequivocal hyperglycemia (plasma glucose > 200 mg/dl) and polydipsia, polyphagia, and polyuria. Type II diabetes, previously known as adult- or maturity-onset, is non-insulin dependent and may require an OGTT for diagnosis. Either type has been described in both children and adults, but Type II usually occurs in overweight adults. Note that the diagnostic criteria for gestational diabetes requires that 100 gm glucose be administered rather than the standard 75 gm glucose load. The terms "potential", "latent", "subclinical" and "pre-diabetes" have been abandoned. Instead "potential abnormality of glucose tolerance" denotes those individuals at high risk of developing diabetes. These persons would include monozygotic twins, siblings or offspring of a Type I diabetic, monozygotic twins or first-degree relatives of a Type II diabetic, women delivering infants weighing over nine pounds, obese individuals, Pima Indians, etc. The prognostic value of repeated OGTTs in these groups is unclear, as at least one study reports the reproducibility of such testing in normal subjects at no more than 50%.<sup>8</sup>

### Etiology

There have been significant advances in our understanding of the probable causes of Type I diabetes although the term "multifactorial" has not been eliminated. A specific etiology rarely can be assigned to an individual case, but the roles of autoimmunity, genetics and viruses are becoming clearer.

**Table 1. Summary of Diagnostic and Classifying Material.**

<b>I. Criteria for Diagnosis</b>	
<b>A. Diabetes Mellitus</b>	
1.	Plasma Glucose $\geq$ 200 mg/dl with symptoms, or
2.	OGTT—a) Fasting plasma glucose < 140 mg/dl and b) 2 hr plasma glucose $\geq$ 200 mg/dl and c) At least one intervening value $\geq$ 200 mg/dl
3.	Fasting plasma glucose $\geq$ 140 mg/dl on more than one occasion
<b>B. Impaired Glucose Tolerance (by OGTT)</b>	
1.	Fasting plasma glucose < 140 mg/dl and
2.	2 hr plasma glucose between 140 mg/dl and 200 mg/dl and
3.	At least one intervening value $\geq$ 200 mg/dl
<b>C. Gestational Diabetes (using 100 gm glucose load)</b>	
1.	Fasting plasma glucose $\geq$ 105 mg/dl
2.	1 hr plasma glucose $\geq$ 190 mg/dl
3.	2 hr plasma glucose $\geq$ 165 mg/dl
4.	3 hr plasma glucose $\geq$ 145 mg/dl
<b>II. Diagnostic Syndromes</b>	
<b>A. Type I Diabetes Mellitus</b>	
1.	Insulin dependent
2.	Ketosis prone
3.	Can occur at any age
<b>B. Type II Diabetes Mellitus</b>	
1.	Non-insulin dependent
2.	Ketosis resistant
3.	Most frequently in adults
4.	Majority are overweight
<b>C. Gestational Diabetes (see above)</b>	
<b>D. Diabetes Associated with Other Disease (Hemochromatosis, chemically-induced hyperglycemia, endocrinopathies, etc.)</b>	
<b>E. Impaired Glucose Tolerance (see above) May worsen, improve, remain stable over time.</b>	

Nearly 80% of all newly diagnosed children with Type I diabetes will have antibodies (ICA) in their sera directed against the islet cell.<sup>10-12</sup> The prevalence of ICA decreases with duration of disease except in those individuals with other associated autoimmune endocrinopathies, such as thyroiditis or Addison's disease.<sup>10</sup> There are at least six different types of ICA classified by the portion of the islet they react with, their ability to fix complement or their immunopathology.<sup>13</sup> The pathophysiologic importance of these antibodies remains unclear; however, at least one type has been shown to be cytotoxic and those which fix complement or react with the surface of the islet cell have been demonstrated in sera of patients prior to the onset of glucose intolerance.<sup>14-15</sup>

An increased prevalence of HLA antigens B8 and BW15 has been detected in Type I diabetics.<sup>12,16</sup> The presence of either antigen increases an individual's relative risk of developing diabetes, while the presence of both B8 and BW15 results in a greater than additive relative risk. HLA B8 and BW15 are in linkage disequilibrium with D-associated antigens DW3 and DW4. Studies in identical twins demonstrate that the possession of both DR3 and DR4

antigens confers a greater predisposition to diabetes than does the possession of either antigen alone.<sup>17</sup> The presence of HLA B-7 or DW2 appears to be protective against the development of Type I diabetes. HLA-identical siblings of Type I diabetic children have a 100 times greater risk of developing the disease than the general population, while the risk in non-identical siblings (no haplotype in common) is not increased.<sup>18</sup>

The clinical evidence that viruses play a role in the etiology of Type I diabetes is somewhat circumstantial and based on seasonal variations in incidence and temporal relationships between the onset of diabetes and certain childhood infections, such as mumps.<sup>19</sup> An association with congenital rubella infections has been described also. Yoon et al have produced diabetes in mice by inoculating the animals with isolates of Coxsackie B4 virus recovered from the pancreas of a child with the acute onset of diabetic ketoacidosis.<sup>20</sup> This is the strongest evidence to date linking viruses to the late development of diabetes in man.

At least one pathogenetic classification of Type I diabetes ties together the roles of autoimmunity, genetics and viruses.<sup>15,21,22</sup> Type Ia insulin-dependent diabetes occurs more commonly in boys who are B8, DR3 and at a young age. This type of diabetes is probably initiated by environmental insults in genetically predisposed individuals. Islet cell antibodies occur but only transiently and are probably a response to islet damage. Type Ib insulin-dependent diabetes occurs more frequently in females and at a later age. This disorder is characterized by lasting humoral and cell-mediated autoimmune features, and patients often have other autoimmune diseases. Although not all Type I patients will fit these categories, these schemes begin to permit the association of certain features of the disease and may eventually aid in developing predictive criteria for both the onset of diabetes and the development of complications.

In recent years it has also become apparent that Type II diabetes is a heterogeneous disorder involving both genetic and environmental factors. The importance of heredity is best illustrated by the nearly 100% concordance for Type II diabetes among identical twins.<sup>23</sup> Studies have also indicated that genes conferring susceptibility to Type II diabetes may be located on the short arm of chromosome 11 in or near the insulin gene.<sup>24</sup>

The importance of environmental factors in the etiology of type II diabetes is illustrated by the dramatic increase in frequency of diabetes in certain isolated populations. For example, 50% of

Pima Indians either have or will develop diabetes.<sup>25</sup> Diabetes was uncommon in these populations until recently, and the increase in frequency cannot be explained by a change in gene pools. Consequently, some change in the environment must have occurred which caused expression of diabetogenic genes which in earlier environments were not expressed or possibly were beneficial.

The past decade has seen a lively debate regarding the relative importance of insulin deficiency and insulin resistance in the pathogenesis of Type II diabetes.<sup>26</sup> This debate is now resolved to the extent that both phenomena are demonstrable and undoubtedly important.<sup>26</sup> It is not yet clear, however, whether insulin resistance precedes and causes insulin deficiency or vice versa. It is possible that both insulin deficiency and insulin resistance are caused by some more fundamental metabolic defect.

## Management

### Diet

Diet is one of the traditional cornerstones of diabetic management. There is no question that dietary factors can contribute to achieving normoglycemia on a metabolic ward. However, in "real world" settings, dietary therapy of diabetes is widely considered to be a failure.<sup>27</sup> The traditional practice has been to assign responsibility for this failure to the patient for failing to follow prescribed diets or heed nutritional advice.

Research in recent years has shown, however, that our knowledge about the role of nutrition in treatment of diabetes is terribly naive. Historically, "diabetic diets" were formulated by assigning specific foods to groups or exchanges based on their composition. The assumption was that isocaloric amounts of food within a group would affect plasma glucose concentrations to the same extent. This assumption was never tested empirically. Results of recent research on this point have been startling. For example, eating ice cream causes plasma glucose to change very little, whereas eating a baked potato causes plasma glucose to rise nearly as much as does ingestion of pure glucose.<sup>28</sup> Even within the same group, foods differ widely. Rice, for example, has relatively little glycemic effect compared to potatoes.<sup>29</sup> One recent report even concludes that adding sucrose to foods has no adverse effect on diabetic control.<sup>30</sup> This observation must be confirmed and extended before applied to routine practice.

The revival of research interest in nutritional aspects of diabetes care has led to several new



practical diets, including high-carbohydrate, high-fiber and very-low-calorie, ketogenic diets.<sup>31,32</sup> At present, it appears that these diets work for some but not all patients, and long-term effectiveness has not been proven for any.

In 1979, the American Diabetes Association published a position statement on diet.<sup>33</sup> This report states that diabetic diets should be nutritious, timed appropriately considering pharmacologic therapy, compatible with individual lifestyles, and aimed at achieving or maintaining normal body weight. It is reasonable for physician and patient, preferably with the assistance of a nutritionist, to try any old, new or even fad diet as long as these four criteria are met and the effect of the diet on diabetic control, assessed by glycosylated hemoglobin, is positive.

### Exercise

Despite the longstanding tradition of recommending exercise for patients with diabetes, the precise benefits and risks of exercise as well as the details of an exercise prescription still have not been documented. However, a strong case can be made for continuing to promote therapeutic exercise if only because it usually enhances physical and psychological well-being and reduces risk for cardiovascular disease. Aerobic exercise, performed for 20 minutes three times a week, has been shown to significantly improve glucose control in children with diabetes.<sup>34</sup> On the other hand, there is evidence which indicates that exercise may actually

increase blood glucose concentrations in patients who are severely hyperglycemic.<sup>35</sup> Obviously, strenuous exercise is contraindicated for patients with severe complications of diabetes.

Until better information becomes available, it is good practice to recommend moderate, individualized exercise programs for patients with diabetes after careful medical evaluation. Patients taking subcutaneous insulin should be warned that absorption of short-acting insulin is markedly increased from depots in exercised limbs. Injection sites should be chosen in areas not being exercised.<sup>36</sup> Patients who perform self-monitoring of blood glucose should evaluate potential beneficial or deleterious effects of exercise on blood glucose control.

### Insulin

The purity of injectable insulin has been greatly improved with the introduction of European products manufactured by Novo Laboratories and Nordisk Industries and the subsequent improvement in purity of Eli Lilly's pure pork insulin (Table 2). These purified products have theoretical applications in the patient with insulin antibodies, insulin allergy, lipoatrophy, lipohypertrophy and marked insulin resistance.<sup>37,38</sup> In addition to the purified pork insulins, human insulin is now available. This product is either synthesized, using recombinant DNA technology or by modification of pork insulin.<sup>39</sup> Preliminary studies suggest that human insulin lowers blood glucose similarly to purified pork insulin

Table 2. Insulins Currently Available (U-100).

Type	Source	Onset	Action Peak	Duration
Short-Acting				
Regular (L,S)	B,B-P,P,H,			
Actrapid (N)	P,H	0.5	2-3	8
Velosulin (ND)	P			
Semilente (L,S)	B-P	1	4-6	12
Semitard (N)	P			
Intermediate-Acting				
NPH (L,S)	B,B-P,P,H			
Protophane NPH (N)	P	2	8-12	12-24
Mixtard (ND)	P			
(30% Reg, 70% NPH)				
Lente (L,S)	B,B-P,P			
(30% Semi, 70% Ultra)				
Monotard (N)	P,H	2	8-12	12-24
Lentard (N)	B-P			
Insulatard (ND)	P			
Long-Acting				
Ultralente (L,S)	B-P	6	14-20	24-30
Ultratard (N)	B			
PZI (L)	B-P,P			

Manufacturer: L-Lilly, N-Novo, S-Squibb, ND-Nordisk Source: B-Beef, P-Pork, H-Human

but with a slightly faster onset and shorter duration of action.<sup>40</sup> Disappointingly, serum antibodies develop to human insulin as they do to pork insulin.<sup>41</sup> Long-term studies are needed to determine the effects of using human insulin initially in newly diagnosed patients on insulin antibody formation and the development of long-term complications.

### Insulin Delivery

In general, there is a trend toward the use of multiple daily insulin injections to treat Type I diabetes. Theoretically, multiple injections of mixtures of short- and long-acting insulins produce multiple insulin peaks which can resemble normal physiologic insulin release. A variety of treatment regimes or algorithms are available.<sup>42</sup> The schedule chosen for a particular patient should be based on the patient's activities, dietary pattern and diurnal glucose values. Multiple injections allow the patient to make choices concerning diet and exercise throughout the day without precipitating extremes of blood sugar.

Perhaps the most impressive development in attempts to deliver insulin in a physiologic manner is the use of continuous subcutaneous insulin-infusion pumps (CSII).<sup>43,44</sup> These systems are designed to deliver a constant basal rate of insulin (approximately 1 unit/hr) while mealtime boluses are adjusted by the patient depending on meal content, anticipated activity and present blood glucose concentration. Experience with these systems has demonstrated their capabilities in normalizing blood glucose and a variety of metabolic abnormalities.<sup>45</sup> CSII appears to be a safe and reliable method of insulin delivery provided both the physician and patient agree to assume the responsibilities entailed. The ADA has recommended that CSII be used only in patients in whom intensive efforts with proper diet and multiple injections fail to achieve an acceptable level of glucose control.<sup>46</sup> These devices should be used in a setting where a trained staff can provide intense education and 24-hour communication service. Several CSII systems are currently available. Since their price and programming capabilities differ, it is important to become familiar with their characteristics before selecting a particular system.<sup>47</sup>

It has become increasingly obvious that no single mode of insulin delivery is appropriate for all patients. This may not be surprising, given the clinical heterogeneity of Type I diabetes. Vigorous attempts to normalize glucose levels in Type I diabetes can be expected to increase the risk of insulin induced hypoglycemia. However, optimal insulin

delivery schemes and appropriate patient responses to changes in diet, exercise and fluctuations in glucose concentrations can reduce this risk. Unfortunately, some diabetics lose the ability to secrete counter-regulatory hormones (glucagon and epinephrine), which are critical to recovery from hypoglycemia.<sup>48</sup> A recent report by White et al describes the use of an intravenous insulin infusion test for identifying those individuals who may be at increased risk of hypoglycemia during intensive therapy.<sup>49</sup>

### Oral Agents

The 1978 VIRGINIA MEDICAL article summarized the University Group Diabetes Program (UGDP) controversy and made specific recommendations for the use of oral hypoglycemic agents.<sup>1</sup> Subsequently, no definitive data on the safety and efficacy of oral agents have appeared, and the questions raised by the UGDP study remain unanswered. The use of oral agents appears to be justified provided that the patient 1) has Type II (non-insulin dependent) diabetes; 2) is not pregnant or lactating; 3) is not under severe physical stress; 4) has no known allergy to sulfonylurea drugs; 5) understands the potential benefits and risks as well as alternative approaches to therapy; and 6) responds to the drugs. Furthermore, it is the responsibility of the physician to monitor glycemic control and to discontinue the drug in the event of secondary failure.

The "second generation" oral hypoglycemic agents, glyburide and glipizide,<sup>50</sup> are likely to become available for use in the United States in the near future. These drugs are sulfonylureas and differ from the first generation agents primarily in potency. Although it has not been proven that the second generation agents are more effective than the first generation agents, these drugs appear to be a useful addition to the pharmacologic therapy of Type II diabetes.

### Self-Monitoring

There is a growing awareness among diabetics and their health care providers that self-monitoring of blood glucose may be the single most important development in diabetes management in decades. Urinary glucose determinations can only reflect previous blood levels, and the quantification of glycosuria has been shown to be fraught with error.<sup>51,52</sup> Knowledge of one's actual blood glucose level gives the patient the information needed to be able to adjust diet, exercise and insulin appropriately. Numerous reports demonstrate that patients can accurately determine their blood concentration us-



Table 3. Systems for Blood Glucose Monitoring.

Visually Interpreted		Test Strips	Range	Cost
Chemstrips bG (Bio-Dynamics)			20-800 mg/dl	\$45/100
Visidex III (Ames)			20-800 mg/dl	\$55/100
Reflectance Meters				
Accu-Check bG (Bio-Dynamics)	Chemstrip bG		40-400	\$150
Beta Scan A (Orange)	Dextrostix		0-396	\$250
Glucocheck II (Larken)	Chemstrip bG		10-400	\$250
Gluco Key (Ulster Scientific)	Dextrostix		10-400	\$225
	Chemstrip bG			
Glucometer (Ames)	Dextrostix		0-399	\$150
Glucoscan II (Life Scan)	Glucoscan		25-450	\$159

ing either visually-interpreted reagent strips or those which require a reflectance photometer.<sup>53,54</sup> Presently available systems are listed in Table 3.

### Glycosylated Hemoglobin

In addition to the new techniques available for patients to monitor their ambient blood glucose levels, commercially available glycosylated hemoglobin (HbA<sub>1c</sub>) assays allow the physician to monitor overall glucose control over a previous 2-3 month period. Hemoglobin A in the presence of glucose undergoes first a non-enzymatic reaction which links the hexose to the amino terminus of the HbA molecule and then an irreversible structural rearrangement to form hemoglobin A<sub>1c</sub>.<sup>55</sup> HbA<sub>1c</sub> levels correlate with 24-hour urinary glucose excretion, are high during acidosis, and low during the remission phase of Type I diabetes. There are several techniques for determining HbA<sub>1c</sub> concentrations; some techniques also may measure an intermediate product which is influenced by acute changes in blood glucose.<sup>56</sup> Hence, unless one is absolutely certain that this labile fraction is removed by the lab prior to assaying for HbA<sub>1c</sub>, only fasting blood samples should be collected. It is important that normal HbA<sub>1c</sub> values for each laboratory be known, as test results vary from one lab or technique to another. HbA<sub>1c</sub> levels may be influenced by HbS or HbF levels.

### Complications

There have been no changes in the reported incidence of diabetic complications. However, it is hoped that the reductions of systolic blood pressure and increases in nerve conduction velocities observed with improved glucose control will be seen in other tissues as well.<sup>53</sup>

Retinopathy can now be detected in its earliest stages by quantitating the amount of injected fluorescein leaking into the vitreous.<sup>57,58</sup> This leakage correlates significantly with glucose control and duration of diabetes.<sup>59</sup> Unfortunately, retinopathy is still being seen in Type I diabetes of short

duration and more frequently in association with hypertension.<sup>60,61</sup>

Evidence has accumulated indicating that glomerulosclerosis is a consequence of hyperglycemia and can be prevented in the laboratory animal by proper glycemic control.<sup>62,63</sup> Unfortunately, renal function in diabetes is abnormal long before the development of clinical nephropathy, and long-term improvement of metabolic control does not reverse nephropathy once present.<sup>64,65</sup> At present there are no markers, with the exception of renal biopsy, for the development of nephropathy prior to the onset of proteinuria and decreased renal function. Treatment of early hypertension, a major risk factor in the development and progression of micro- and macro-angiopathy, has been shown, however, to inhibit progression of diabetic nephropathy.<sup>66,67</sup>

### Psychological Aspects

Diabetes mellitus is a life-style disease. Regardless of the type, etiology or treatment plan, diabetes becomes a part of everyday life. Incorporation of good self-management practices into everyday activity is necessary to attain and maintain good glucose control. Diabetes is the only chronic disease in which the patient is given the bulk of the responsibility for medical observation and decision-making. The pressures on parents, family members and patients vary but are based on a host of social and psychological factors. Anxiety, anger, resentment and fear are common, as are overprotection and denial.<sup>68-70</sup> It is important for health care providers to seek out social and psychological information and to assist patients in dealing with their disease. It is encouraging to note that intensification of efforts to attain improved diabetic control has been associated with positive psychosocial benefits in both adolescents and adults.<sup>71,72</sup>

### The Future

Fascinating developments in the understanding and treatment of Type I and Type II diabetes continue to be reported at a rapid rate. Speculation

suggests that this review will be outdated long before the previous report.<sup>1</sup> Scientists are now studying the use of immunosuppressive agents to prevent B-cell destruction prior to the onset of overt hyperglycemia in Type I diabetes. Recombinant DNA techniques are being developed to identify genetic markers for Type II diabetes. The National Institutes of Health are sponsoring a ten-year controlled study of the role of glucose control in the development of long-term complications in 600 Type I patients. Human islet cell transplantation is scheduled to begin in the near future. It is anticipated that within a decade, novel therapeutic choices will be available to the physician caring for patients with diabetes and that these treatment plans will help achieve an improvement in both the mortality and morbidity associated with this disease.

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## BOOKS

**Progress in Clinical Pathology, Volume IX.** Edited by Mario Stefanini, MD, Fred Gorstein, MD, and Louis Fink, MD. New York, Grune & Stratton, 1983, 288 pp, illustrated, \$64.50.

This is the ninth volume in a widely known and respected series designed to give the reader a "state of the art" look at clinical pathology. The first volume was published in 1966. Dr. Stefanini, pathologist at Humana Hospital Clinch Valley, Richlands, Virginia, was the sole editor of Volumes I-VII. For Volume VIII he solicited the partnership of Dr. Ellis Benson of the University of Minnesota, and Dr. Gorstein and Dr. Fink joined him for this University of Vanderbilt issue.

I highly recommend this book. It is an extremely current and well-organized overview of the progress in clinical pathology. In a field that is becoming increasingly complex, as well as one in which significant advances in understanding disease are being made, this book provides critical information. While the chapters titles represent the interest and expertise of the Vanderbilt University contributors, they well reflect current developments in the field.

There is much information for anyone with knowledge of the discipline. However, a note of caution: While each of the chapters may serve as a reference for its title, the information is not for the novice. Basic knowledge is presumed, thus making this an informative presentation, without dilution, for the knowledgeable.

The book begins with an up-to-date presentation of the coagulation pathways and the evolving role of tissue factor in initiation of the reaction. Clinical considerations and their importance are given.

This is followed by an outstanding presentation of

fibrinogen and its derivatives. The format of basic biochemistry, pertinent assays, products and clinical relevance is followed. It is an excellent discussion of the pivotal role of fibrinogen in humoral and cellular hemostatic mechanisms.

These high standards of presentation are continued throughout subsequent chapters dealing with such diverse subjects as blood component therapy, susceptibility testing, interferon, monoclonal antibodies and estrogen receptor analysis.

This reviewer's personal bias and interest are evident in his admiration for the chapters on flow cytometry, non-Hodgkins lymphomas and B-cell neoplasms. These are the high points of this volume. The consideration of flow cytometry is an excellent presentation of the current application of cell sorting and monoclonal antibody application keynoting the technical applications that may be expected in the study of neoplasia and the neoplastic disorders. Clearly, this is a "now" technique rapidly approaching routine assay status.

The chapter on blood and bone marrow involvement by non-Hodgkins lymphoma is a concise, informative, yet comprehensive treatment of the topic. As would be expected, the classification nomenclature of Lukes and Collins is utilized. However, any possible confusion is dispelled by the outstanding illustrations that are of such excellent quality there can be little doubt of the morphological characteristics of the disorders. I have rarely seen such high quality black and white photography of the malignant lymphomas.

The book begins on a very high plane, which continues throughout. It was a pleasure to review because of the excellence of its organization, the timeliness of its material, and the outstanding illustrations. This reviewer was left not only satisfied but smarter.

CHARLES L. JOHNSTON, JR., MD

Box 597, MCV Station  
Richmond VA 23298



# Fatal Typhlitis Secondary to Procainamide-Induced Agranulocytosis

Richard M. Clary, MD,  
Wyatt S. Beazley III, MD,  
and Robert W. Henley, Jr., MD,  
*Richmond, Virginia*

**T**YPHLITIS, or inflammation of the cecum, is a frequently described complication of hematologic malignancies. The typical patient is a young child with leukemia undergoing chemotherapy. More rarely, neutropenic adults presenting with typhlitis have been reported.<sup>1,2</sup> The vast majority of these have been in patients with leukemias. Ryan and Morrissey recently reported a case of necrotizing colopathy of the cecum in a patient with drug-induced agranulocytosis.<sup>3</sup> We have recently seen a similar patient presenting with fatal typhlitis.

## Case Report

A 74-year-old white female was admitted with chief complaints of fever, sore throat, abdominal pain and diarrhea. She had a history of paroxysmal atrial fibrillation and had been maintained on propranolol 20 mg po tid and digoxin .25 mg q day for over two years. In addition, she had been taking aspirin 5 grains per day following an episode of possible transient cerebral ischemia two years previously, and Transderm®-Nitro, a 5-mg patch q day, for stable Class II angina. Two months prior to admission she had been hospitalized with recurrent atrial fibrillation and had converted to sinus rhythm with the addition of sustained release procainamide (Procan®-SR) 750 mg po q 6 h to her usual regime. She did well until twelve days prior to admission when she developed fever and sore throat with malaise. Symptoms persisted despite outpatient treatment with po amoxicillin. Two days prior to admission she developed abdominal pain and diar-

rhea. Because of persistence of symptoms she was seen in the emergency room and admitted.

Examination revealed a temperature of 100.2 degrees with a blood pressure of 140/80 and a regular pulse at 100 per minute. There was a white exudate in her throat, but KOH prep was negative. Abdominal exam showed marked right lower quadrant tenderness with question of rebound. There was a fullness in the right lower quadrant to palpation. Bowel sounds were present. There was no organomegaly. Rectal showed non-specific tenderness with guaiac-negative stool.

Laboratory showed hematocrit of 34%. Total white blood count was 200/mm<sup>3</sup>, 100% lymphocytes with increased platelets and no neutrophils. Blood chemistries were unremarkable. Bone marrow was hypocellular with no granulocytes seen. There were increased megakaryocytes. Gram stain of the stool showed normal flora.

The patient was cultured thoroughly and placed in reverse isolation. She was begun on intravenous tobramycin and clindamycin and nystatin (Mycostatin®) by mouth. Procan-SR was discontinued. Clostridial toxin was negative. ANA was positive at 1:320 dilution with a hemogenous pattern. CT scan showed a 7.5-cm soft tissue mass in the right lower quadrant, felt probably to be right colon. Gallium scan showed focal increased activity in the right lower quadrant. Temperature spiked to 102 degrees. Although there had been no improvement in the hematologic picture it was deemed mandatory to explore the patient at this time to rule out a source of peritonitis in the right lower quadrant. Operation revealed a markedly edematous beefy red cecum. The cecum was excised, with an end ileostomy and ascending colon mucus fistula being performed. Pathology report showed extensive mucosal ulceration with superficial invasion by *Candida*. Culture of peritoneal fluid revealed no growth.

On the second postoperative day, the white count began to rise and was 21,000 by the fifth postoperative day. The patient progressed well until postoperative day #10, when she complained of shortness of breath and arrested. Attempts at resuscitation were unsuccessful. A post-mortem examination was not performed.

## Discussion

The agranulocytosis in this patient was almost certainly secondary to procainamide, which had been started two months previously. This is a recognized, although rare, complication.<sup>4</sup> A recent report by Berger describes the occurrence of agranulocytosis following treatment with the new sus-

Address correspondence to Dr. Clary at 425 North Boulevard, Richmond VA 23220.

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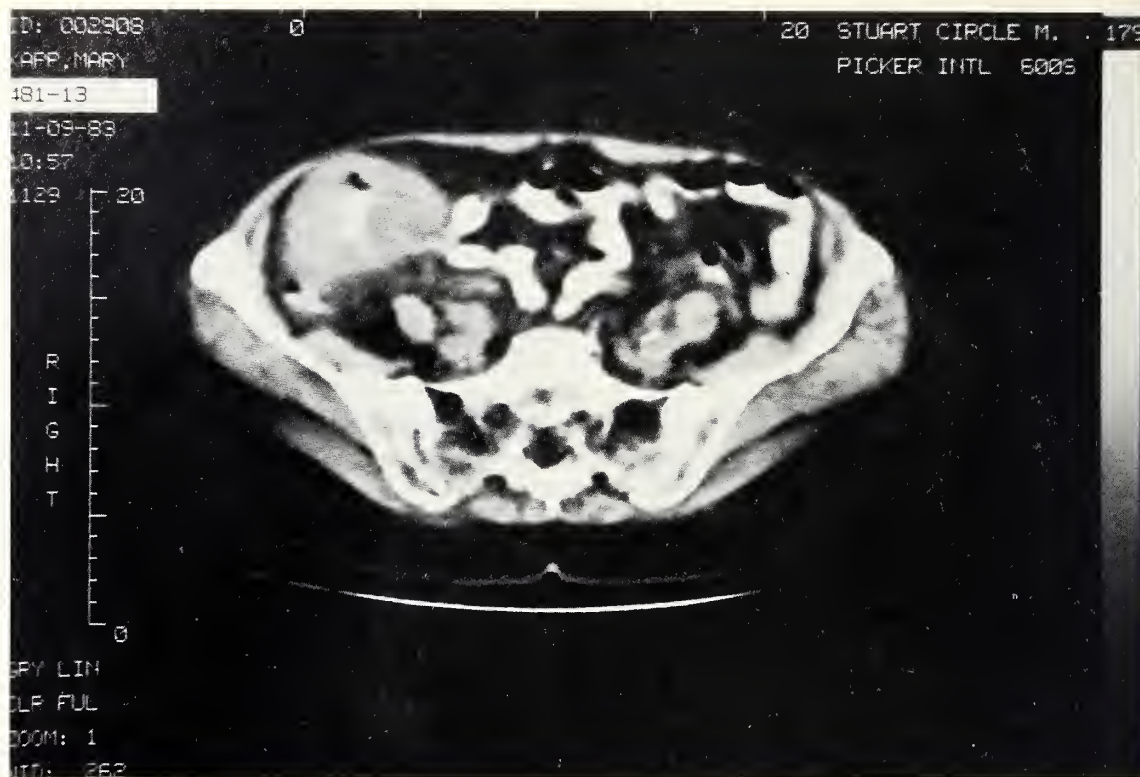


Fig. 1. CT scan revealed 7.5-cm soft tissue mass.

tained-release form of procainamide.<sup>5</sup> The mechanism of this agranulocytosis is unknown. The positive ANA in our patient suggests an autoimmune mechanism, but this has not been a constant finding in procainamide-induced agranulocytosis. Mortality has been reported as 24%.<sup>4</sup>

While immunosuppressed with neutropenia, the patient developed signs and symptoms of an acute abdomen. These are classic findings in necrotizing cecitis or typhlitis. Although usually seen in patients with hematologic malignancies, our case and the case reported by Ryan and Morrissey confirm that any patient with agranulocytopenia may be at risk for typhlitis. Although multiple etiologies have been suggested, we agree with Ryan's suggestion that bacterial invasion through a mucosal defect is the most likely. Without normal host defenses, this results in the life-threatening infection described.

The best management of typhlitis is unknown, with some reports of recovery despite conservative management. Unfortunately, the diagnosis is seldom made preoperatively and in most patients the differential includes definite surgical lesions. Because of the similarity to appendicitis, confusion may delay surgical consultation in patients known to have had previous appendectomy. As the condition may progress to frank rupture, we feel our

operative therapy was warranted. The sudden death of our patient despite hematologic recovery was most likely secondary to pulmonary embolus or acute myocardial infarction.

Inflammation of the cecum in the immunosuppressed patient remains an elusive but life-threatening complication. Although surgery may be associated with high morbidity and mortality, observing a compromised patient with signs of an acute abdominal infection appears the greater evil. Granulocytopenia from any cause may predispose a patient to typhlitis.

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# Mitral-Valve Prolapse Presenting with Seizures: Case Report

Thomas T. Smirniotopoulos, MD,  
*Ranson, West Virginia,*  
and George L. Sheppard, MD,  
*Winchester, Virginia*

**M**ITRAL-VALVE PROLAPSE (MVP) is a common disorder, occurring in approximately 6% of otherwise healthy young female patients.<sup>1</sup> The usual presenting symptoms include atypical chest pain, palpitations, dyspnea and light-headedness. We describe a patient with previously asymptomatic MVP who presented with new onset of seizures.

## Case Report

A 34-year-old white female presented to Jefferson Memorial Hospital emergency room with new onset of seizures. The patient had previously been in good health and was on no medications. The event was witnessed by the patient's husband, who described generalized tonic-clonic seizure activity of several minutes' duration.

Upon arrival at the hospital the patient was alert and oriented. Blood pressure was 100/80 with a pulse of 88 and a respiratory rate of 16. She was afebrile.

Examination of the head revealed normal pupillary reaction to light. Fundoscopic examination demonstrated sharp disk margins. There were no cranial nerve deficits. There were lacerations of the tongue. The neck was supple and the carotid pulses were normal. There were no carotid bruits.

Auscultation of the lungs revealed normal breath sounds. Auscultation of the heart was remarkable for a loud mid-systolic click with a grade II/VI late

From the department of emergency medicine, Jefferson Memorial Hospital, Ranson, West Virginia (Dr. Smirniotopoulos), and the department of neurology, Winchester Memorial Hospital, Winchester, Virginia (Dr. Sheppard). Address correspondence to Dr. Smirniotopoulos at his present place of practice: 2817 Duke Street, Alexandria VA 22314.

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systolic murmur. The remainder of the physical examination was within normal limits.

The patient was transferred to Winchester Memorial Hospital for further evaluation. Following a second seizure she underwent an electroencephalogram, which showed intermittent bursts of mixed theta and delta slowing but no evidence of seizure activity. Computed tomography of the head was normal. Lumbar puncture revealed normal cerebral spinal fluid.

A 12-lead electrocardiogram and 24-hour Holter monitor were within normal limits. Because of the presence of the click-murmur, the patient underwent M-mode and 2-D echocardiography. MVP was documented by the presence of hammocking of the mitral valve in late systole (Fig. 1).

The patient was started on carbamazepine 200 mg tid, dipyridamole 25 mg qid, and aspirin 10 grains bid. She was discharged on the third day and is symptom-free as of this writing.

## Discussion

Complications of mitral-valve prolapse are rare and include progressive mitral regurgitation, spontaneous rupture of the chordae tentonae, bacterial endocarditis, and arrhythmias, including ventricular fibrillation.<sup>2</sup> In 1980 Barnett et al<sup>3</sup> reported an increased prevalence of MVP in young patients with cerebral ischemic events. Among 60 patients under 45 year of age they found a 40% prevalence of MVP, as compared to 6.8% in age-matched controls. In six with transient ischemic attacks (TIA) and 12 with cerebral infarct (CI), mitral-valve prolapse was the only predisposing factor.

Other investigators have found a similar associa-

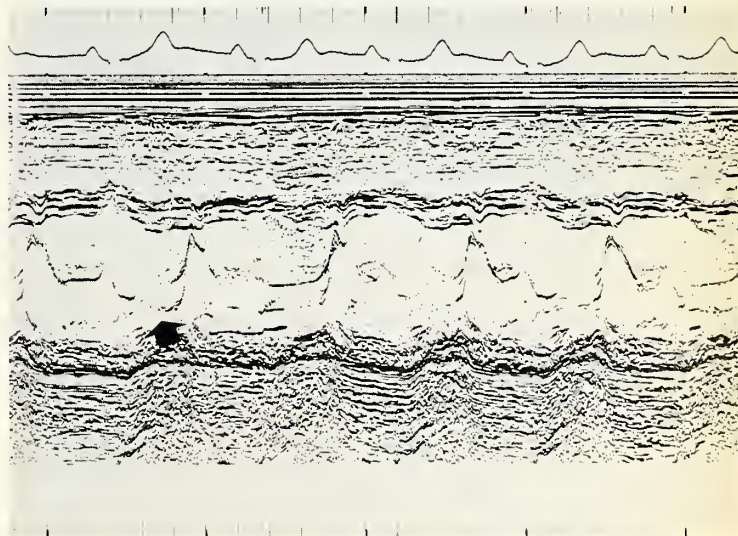


Fig. 1. Echocardiogram shows hammocking of the mitral valve in late systole (arrow).

tion between MVP and cerebral ischemic events in young adults. Hanson et al<sup>4</sup> studied 24 patients with cerebral ischemic events and MVP in whom no other predisposing factors were found. The average age was 42.6 years for 18 women and 45.0 for six men. Only four of these patients had documented MVP prior to the ischemic event. Two patients presented with seizures, one had retinal artery occlusion, six had TIA and 15 had CI.

Jones et al<sup>5</sup> described 43 patients presenting with cerebral embolus who underwent echocardiography. Nine were found to have MVP (21% prevalence). Two of these presented with TIA and seven with CI. Sandok and Giuliani<sup>6</sup> described a four-fold increased incidence of cerebral ischemic events in 1,138 patients with documented MVP. The average age of these patients was 48.4 years. Two had retinal artery occlusion, two had amaurosis fugax, three had TIA, and 33 had CI.

Most investigators agree that patients with MVP who have had symptoms of cerebral ischemic events should be treated prophylactically with antiplatelet drugs, including aspirin and dipyridamole.

Our patient is within our knowledge the third reported case of MVP presenting with seizures. The actual incidence may be higher because MVP has not previously been considered a risk factor for seizures and thus may not have been looked for. We recommend echocardiography in young adult patients who present with new onset of seizures (without obvious neurologic etiology) to rule out the presence of MVP. In those patients found to have MVP, anti-platelet prophylaxis should be added to the usual anti-convulsant regimen.

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## Ultrasound Imaging in Massive Ovarian Edema: Case Report

P. M. Fitzer, MD,  
*Newport News, Virginia*

**M**ASSIVE ovarian edema (MOE) is a recently recognized clinical syndrome in young women.<sup>1,3</sup> Marked enlargement of an ovary is present, sometimes accompanied by virilisation. Only three examples of sonographic imaging of the syndrome have been presented.<sup>4,5</sup> This report notes an additional case of MOE with similar sonographic findings.

#### Case Report

A 16-year-old nullipara was hospitalized with acute right lower quadrant pain. She was afebrile; there was slight lower quadrant tenderness, but no mass was noted. The white blood count was mildly elevated but was normal the next day when she became asymptomatic and started a normal menstrual period.

On a routine visit four weeks later a suprapubic mass was palpated; she was still asymptomatic, and a pregnancy test was negative. Sonography revealed a 10 × 5 cm pelvic mass on the right side, just cephalad and separate from the normal uterus; the mass was predominantly sonolucent (Fig. 1). The images suggested a right ovarian origin, possibly hemorrhage into a cyst.

At laparotomy the right ovary was replaced by a pinkish gray, rubbery ovoid 12 × 6 cm solid mass; the left ovary had a similar appearance and measured 5 × 6 cm. The right ovarian mass was removed, and a wedge resection of the left ovary was done.

From the Department of Diagnostic Radiology, Medical College of Virginia/Virginia Commonwealth University, and the Department of Radiology, McGuire Veterans Administration Medical Center, Richmond. Address correspondence to Dr. Fitzer at 501 Riverside Drive, Newport News VA 23606.

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Pathological examination of both ovarian specimens showed a well vascularized, markedly edematous stroma, with occasional foci of more condensed normal appearing stroma; no areas of hyperthecosis were present. The final diagnosis was bilateral massive ovarian edema.

Postoperatively, the patient has remained infertile, in spite of normal menstrual periods.

## Discussion

Massive ovarian edema typically occurs in adolescents.<sup>1,3</sup> Most patients present with pain and an adnexal mass, but virilisation alone may be noted. Only one ovary is involved and intermittent or partial torsion of the ovarian pedicle is usually implicated; impairment of ovarian perfusion may be responsible for abnormal steroid secretion and virilisation. This might account for the infertility experienced by the present patient. Clinically, this patient is most unusual as the pathologic changes involved both ovaries without obvious torsion or any other cause of abnormal circulation.

The ultrasound images are not diagnostically specific. A well circumscribed predominantly sonolucent pelvic mass may be due to hemorrhagic cyst, ovarian neoplasm, pelvic inflammatory disease or appendiceal abscess. The sonolucent texture of the present case is quite similar to the three previously reported examples of MOE.<sup>4,5</sup>

The diagnosis of massive ovarian edema should be considered in an adolescent girl presenting with a rapidly enlarging sonolucent adnexal mass, especially if virilisation is present.

The author thanks William M. Bangel, MD, for his help in preparation of this manuscript. The secretarial assistance of Paula Kornegay and Mary Diggs is greatly appreciated.

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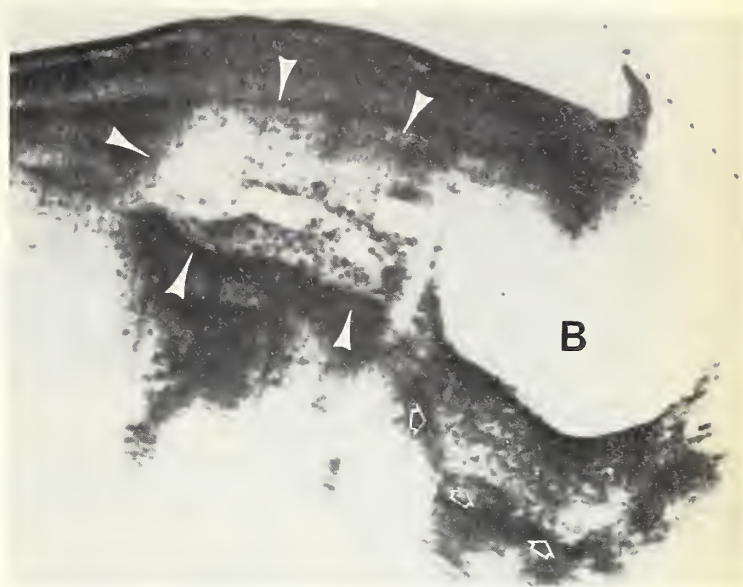


Fig. 1A. Sagittal midline image. Mass (small arrows) cephalad to filled bladder (B), separate from normal uterus (open arrows).

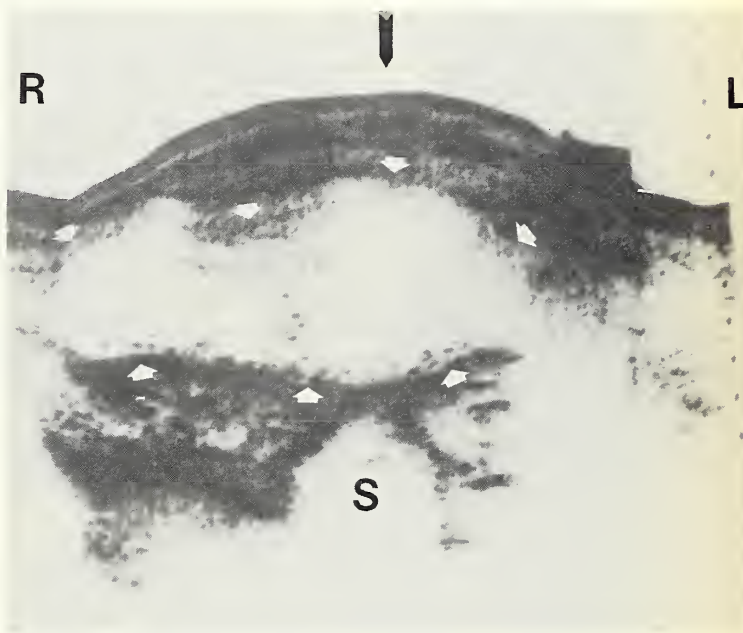
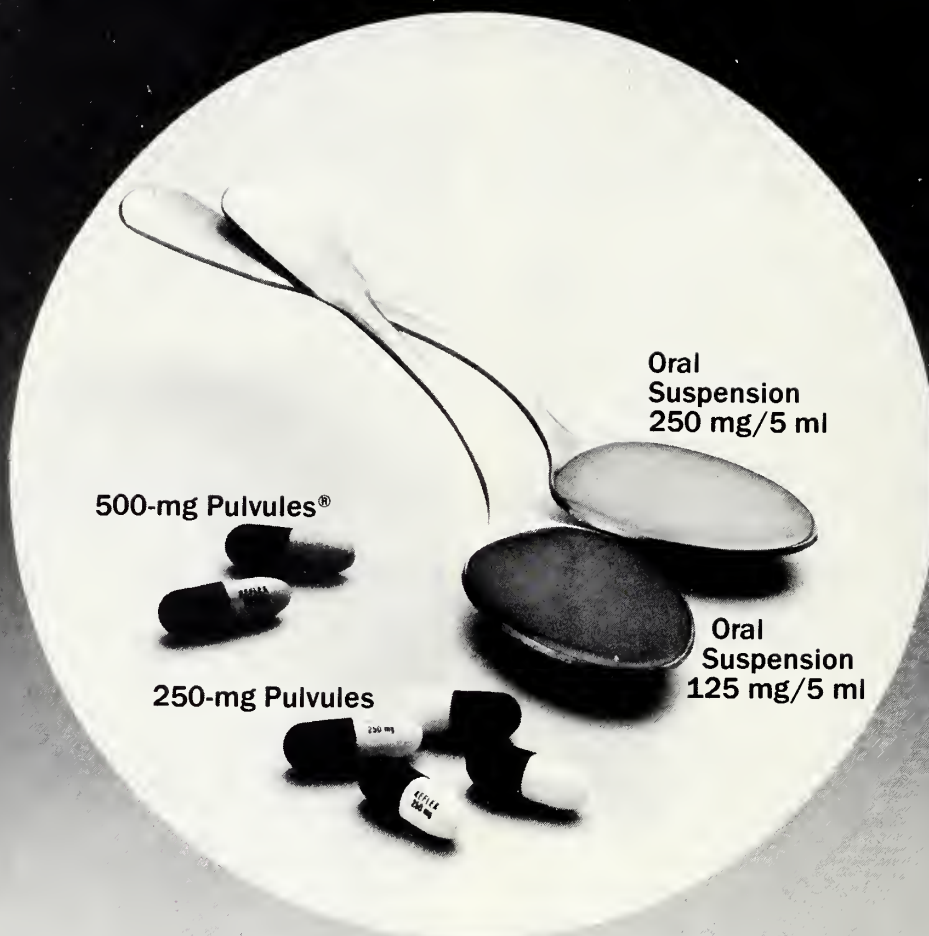


Fig. 1B. Transverse image cephalad to bladder. Mass (small arrows) predominately sonolucent, with scattered low level echos. Black arrow = midline; S = spine.

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# VIRGINIA MEDICAL

## EDITORIAL

### Immunization Crisis

**W**HEN did you last see a case of diphtheria? There were only five cases reported in the United States in 1983.<sup>1</sup> How often do you see (or hear of) tetanus, pertussis, measles, mumps or rubella? One of the most effective of all approaches to the control of disease is that of the routine (required) immunization of young children. But there is trouble.

Physicians have long since become painfully aware of the litigious nature of our present-day society and the resultant high cost of medical malpractice insurance. Pharmaceutical companies have had a like experience in regard to medical liability, and a recent court case has resulted in a decision affecting the availability of vaccines for the immunization of young children.

DTP (diphtheria/tetanus toxoid and pertussis vaccine) has been successfully utilized for many years. While it is recognized that pertussis vaccine is the least effective of the three and is more likely to be the cause of untoward reaction, use of the vaccine has markedly reduced both the incidence and the mortality of this disease.

All DTP vaccine in the U.S. has been manufactured by three pharmaceutical companies: Eli Lilly & Company, Wyeth Laboratories, and Connaught Laboratories. Because of recent medical liability actions, Wyeth Laboratories has withdrawn from

the field and will no longer manufacture DTP vaccine. Both Lilly and Connaught plan to increase production, but there is already a shortage of vaccine in some areas of the country. Further, the cost of available vaccine has risen precipitately. A 15-dose vial of DTP vaccine available to the physician one year ago for \$4.50 is now \$42—almost a ten-fold increase. It should be noted also that six to eight years ago there were four producers of poliomyelitis vaccine; today, the vaccine is made available by only one company.

The American Academy of Pediatrics, in an attempt to solve the problem, is promoting the concept of no-fault insurance to be financed by the U.S. government and paid to the parents of any child injured by immunization through no fault of the physician or the manufacturer. Legislation has been introduced in both the Senate and the House of Representatives, but as this was written, there seemed little likelihood of passage during the 1984 session of Congress.

What else can the physician do? It is incumbent upon him/her to follow certain rules in order to minimize the possibility of an untoward reaction. As listed by the Committee on Infectious Diseases, American Academy of Pediatrics, these are as follows:

“Pertussis immunization is contraindicated for



those who have any of the following reactions after administration of a pertussis-containing vaccine: (1) a severe neurologic reaction; (2) persistent, unresolvable screaming for three hours or more; (3) a hyporesponsive, shock-like state; (4) temperature of 40.5°C (105°F) or greater, unexplained by another cause within 24 hours following immunization; (5) a convulsion within 48 hours following immunization; or (6) an allergic reaction to the vaccine."<sup>2</sup>

The problem is not likely to be solved until no-

fault insurance is enacted into law. However, physicians must try in every way to be sure that no-fault insurance means just that—no fault.

EDWIN L. KENDIG, JR., MD

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## New Approaches to Diabetes

**I**N THIS ISSUE of VIRGINIA MEDICAL Clarke and Pohl present a summary article on recent advances in diabetes mellitus. Attention is given to the substantial amount of important new information arising from basic research in the field, but the major thrust of their presentation is the clinical evaluation and management of patients with glucose intolerance. Although the basic tenets of diabetic management remain the same, increased emphasis on stringent control of blood glucose and newer types of insulin and methods of delivering insulin have emerged. These innovations have altered the approach to treatment of insulin dependent (Type I) diabetics.

With increasing frequency we are told that tight blood glucose control is good and loose control is bad for the diabetic, and a massive educational program has been launched informing both patients and physicians of the benefits of better control. The non-insulin-dependent (Type II) diabetic has also been encouraged to lose more weight, eat high-fiber foods and exercise more to keep blood sugar within

the normal range. Weight reduction in this group is uniformly accompanied by improved glucose control. Home glucose monitoring has enhanced interest and cooperation, while giving the patient a sense of active participation in regulating his diabetes. Glycosylated hemoglobin (Hgb Alc) continues to be useful in assessing long-term diabetic control.

Better control seems to be worth the effort. Although the jury is still out, evidence is accumulating to indicate that closer regulation of blood glucose has not only immediate advantages but also improves the chances of avoiding at least some of the chronic complications of diabetes.

For VIRGINIA MEDICAL's readers, Clarke and Pohl have summarized a vast literature on these subjects in a timely, concise and uncommonly readable manner. A reprint of their paper would make a handy and valuable attachment to a favorite general medical text.

HERSCHEL L. ESTEP, MD

Eastern Virginia Medical School  
150 Kingsley Lane, Norfolk VA 23505

## Politics—As Usual

**D**URING the past session of the Virginia General Assembly, The Medical Society of Virginia tried a new approach. During each day on which the Assembly was in session, designated physicians were always present. They were not there to act as lobbyists, but to be a source of information to the legislators. Because of the geographical location,

members of the Richmond Academy of Medicine constituted the majority of those attending. The success of the operation dictates that the project will be repeated in 1985. Volunteers are encouraged.

E.L.K., JR., MD

# HISTORICAL NOTES

## Nathaniel Chapman: First President of the American Medical Association

**I**N the list of American Medical Association presidents, the name of a native Virginian leads all the rest: Dr. Nathaniel Chapman, who in 1847 became the fledgling organization's first president. True, Dr. Chapman was then an illustrious professor and editor in Philadelphia, but he had been born, schooled and apprenticed to medicine in Virginia.

His professorship was the prestigious Chair of the Theory in Practice of Medicine and Clinical Medicine at the University of Pennsylvania. As an editor, he founded in 1820 the *Philadelphia Journal of the Medical and Physical Sciences*, which is still published today as the *American Journal of Medical Sciences*; it is the oldest existing monthly medical journal in this country and the second oldest journal in the English language.<sup>1,2</sup> (The *Edinburgh Medical and Surgical Journal* preceded it by 15 years.)

Dr. Chapman was born on May 28, 1780, in Fairfax County. His early education was in the "common" schools in the neighborhood of Summerhill, Virginia, and at the Academy in Alexandria founded by George Washington. His medical apprenticeship began in the Georgetown office of Dr. John Weem and continued in Alexandria, where he studied with Dr. Elisha Dick.<sup>1,3</sup>

When he was 17, Chapman moved to Philadelphia to become a student at the University of Pennsylvania and a private pupil of Dr. Benjamin Rush. At graduation in 1801 his doctoral thesis was on "Hydrophobia," written in answer to an attack made on Rush's views on the subject.<sup>2</sup> After graduation, he traveled to Europe to continue his medical studies, spending the first year in London as a pupil of John Abernethy and the next two in Edinburgh.

Returning to Philadelphia in 1804, he began his teaching career at the University of Pennsylvania, initially presenting obstetrical lectures. Nine years later he was elected to the Chair of Materia Medica, which he held for two years. Of this tenure, Jackson writes in his biographical sketch,<sup>4</sup> "It is my impres-

sion that the courses delivered by him . . . were in advance of those of his predecessor."

Chapman's lectures were published in 1817 in a text entitled "The Elements of Therapeutics and Materia Medica." It was the first systematic treatise on materia medica and therapeutics produced in this country,<sup>5</sup> and presented the concept that relating materia medica to the practice of medicine was indispensable to the rational treatment of diseases. Substances within the materia medica, according to Chapman, had to be studied to determine their capacities for action on the host, their influence on the host's vital functions, and their effect on the disease state of the host.<sup>4</sup>

Following the death of Professor Benjamin Smith Barton in 1816, Chapman was elected to the chair he held when he was elected the first AMA president. He held this position until 1850, exercising through his students a far-reaching influence on American medical practice.

**T**HE MEDICAL THEORIES proposed by Chapman, like those of his contemporaries, were usually based on a matter of opinion rather than scientific proof. He wrote that "the operation of medicine does not depend on any of the common laws of matter but on a principle incident to vitality alone. Of the various doctrines of vitality, one only appears to me to be at all well-founded. It presumes that every animated body, animal or vegetable is endowed with a primordial principle of life."<sup>3</sup>

Chapman's central medical theme was that there was a profound influence by the stomach upon the rest of the body. He wrote: "Conveniently situated for the purpose, the stomach is probably the throne of a vital principle from which would seem to emanate any influence that, diffused over the system, preserves the order of the parts, and sustains the vigor, tone and well-being of the whole animal economy. Assaulted, however, by the impressions which it cannot resist, this organ as the center of



association becomes the seat of the first link in the chain of most diseases, and is always the chief medium of the operation of our remedies, in the correction of morbid arrangement".<sup>3</sup> He further proposed "no viscus or organ, not even the brain itself, can be compared to [the stomach] in this respect of which occupies so important a station in the animal economy".<sup>4</sup>

He objected to the theory that medicines were absorbed into the blood and there performed their actions. He theorized that the action of medicine was due entirely to the impression the medication made upon the stomach.<sup>3</sup> For example, Peruvian bark cured malaria by the sharp and peculiar blow it dealt the stomach.

The importance given the stomach by Chapman was also found in his concepts of the etiology of diseases. Fever was considered a disease caused by local irritation, usually located in the stomach.

Chapman taught, reports Jackson,<sup>4</sup> that the various organs and systems were associated in health, and in disease acted and reacted on each other. Chapman termed this agent of association actions "sympathy" and wrote: "It must be confessed, at present we have no very distinct intelligence relative to [sympathy's] nature".<sup>4</sup>

Chapman was one of the great American medical editors, writes Eckman.<sup>8</sup> As early contributors to his infant journal he drew Philip Syng Physick, W. P. Dewees, Charles Caldwell, J. R. Coxe and W. E. Horner, with their help attempting to advance medical knowledge among the post-colonial practitioners. During this era there was ill-will between American and English medical literature, and Chapman took an active role in sending barbs across the Atlantic.

**T**HE American Medical Association, first called the National Medical Association, grew out of a convention in New York in 1846 which was held at the invitation of the New York Medical Society.<sup>6</sup> Its first organizational meeting occurred the next year in Philadelphia, and it was then that Dr. Chapman was elected the first president. In an address in Baltimore in 1848 Chapman said, "With the present century, the spirit of philosophy began to be infused into [medicine], creative of real and substantial improvements in its theories and modes of practice, raising it from a low and conjectural art, to a place among the legitimate sciences, by which great good is already attained, and further benefit of inestimable value and promised to suffering humanity".<sup>7</sup>

Among Dr. Chapman's other major accomplish-

ments was the presidency of the American Philosophical Society, the learned society founded in 1743 by Benjamin Franklin. The list of physicians who have been members of this society comprise a true hall of fame of American medicine and includes such luminaries as Thomas Cadwalder, Harvey Cushing, Simon Flexner, Samuel D. Gross, I. Minis Hays, Chevalier Jackson and William Edmund Horner.

Dr. Chapman was also president of the Philadelphia Medical Society and a member of the College of Physicians of Philadelphia. He founded in 1817 the Philadelphia Medical Institute and for 25 years delivered there a summer course of lectures. He gave clinical lectures at the Hospital of Philadelphia Almshouse and was co-director, with Dr. Thomas Hewson, of the City Yellow Fever Hospital. The citizens of Philadelphia presented him with a massive silver pitcher in gratitude for his valuable services on their behalf during the terrible cholera epidemic of 1832.<sup>9</sup>

It is written of Dr. Chapman that if he "had a greeting more cordial, or a grasp of the hand more friendly and earnest than another, it was reserved for such of his pupils as were from his native state,



NATHANIEL CHAPMAN (1780-1853)  
*Pen and ink drawing by Mark D. D'Amato*

Virginia, as he ever retained and manifested a decided preference for Virginia and everything Virginian".<sup>9</sup>

In the spring of 1850, because of declining health, Dr. Chapman resigned his professorship at the University of Pennsylvania and was elected an emeritus professor by the board of trustees. His health continued to fail and he died on 1 July 1853.

NICHOLAS A. D'AMATO, MD

Department of Pathology  
DePaul Hospital  
150 Kingsley Lane, Norfolk VA 23505

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### In Chapman's footsteps

Four Virginia physicians have served the American Medical Association as President, most recently Dr. Richard E. Palmer of Arlington, who is shown in the photograph above as he delivered the Presidential Address at the AMA meeting in San Francisco in 1977.

The other three are Beverley R. Wellford, Fredericksburg, elected in 1852; Dr. Hunter Homes McGuire, Richmond, 1893; and Dr. Walter B. Martin, Norfolk, 1952. All had previously served as President of The Medical Society of Virginia.



# VIRGINIA MEDICAL OBITUARY

## W. Callier Salley, MD

Dr. W. Callier Salley, who from 1965 to 1969 was Speaker of The Medical Society of Virginia's House of Delegates, died August 16 at Walter Reed Convalescent Center in Gloucester at the age of 79. He had practiced in Norfolk for 26 years before moving to Gloucester County five years ago.

Born in Fort Deposit, Alabama, Dr. Salley received a bachelor's degree from the University of Alabama, a doctorate in romance languages from the University of North Carolina, and his degree as doctor of medicine from the University of Georgia. He first was a teacher, with posts at colleges in Georgia and Ohio, before entering the medical profession as an internist specializing in diabetes.

He had belonged to The Medical Society of Virginia for almost 40 years and was a long-time member also of the American Diabetes Association. He belonged previously to the Norfolk Academy of Medicine but on moving to Gloucester joined the Mid-Tidewater Medical Society.

## A. W. E. Bassett III, MD

Dr. Andrew W. E. Bassett III, founder of the Peninsula Medical Society in Hampton, died September 5 at his Hampton home. He was 62.

Born in Hampton, Dr. Bassett was graduated from Virginia Union College in 1940 and from the Howard University College of Medicine in 1944. He was a past president of the Old Dominion Medical Society and belonged also to the American Society of Abdominal Surgeons.

## J. Glenn Cox, MD

Dr. J. Glenn Cox, Hillsville, died July 31 at the Lewis-Gale Hospital, Salem, at the age of 83. Ill health had forced his retirement after serving Carroll County residents for 42 years.

Born in Carroll County, Dr. Cox was educated at William and Mary, then, in 1922, embarked on the study of medicine at the Medical College of Virginia. He contracted tuberculosis and returned to Hillsville for a time, but resumed his medical studies in 1928 and received his degree. He worked first

for the State Department of Health, then established his general practice in Hillsville.

Dr. Cox was Carroll County coroner for a number of years and during World War II was medical examiner for the selective Service Board.

He had belonged to the Southwestern Virginia Medical Society and The Medical Society of Virginia for 53 years.

## J. T. Showalter, MD

Dr. Josiah T. Showalter, long-time Christiansburg physician, died July 30 at the age of 72. He had diagnosed himself as having lymphatic leukemia last October but continued to see patients in his office until April.

A native of Cambria, Dr. Showalter was the son of Dr. A. M. Showalter, who began Altamont Hospital in Cambria. "Dr. Joe," as the son was known, was graduated from Virginia Tech and received his medical degree in 1936 from the University of Virginia School of Medicine. During a six-year military career, he served at hospitals in this country and the Pacific. Returning to Montgomery County after World War II, he worked with his father until 1971 and then began a family practice in Christiansburg with the late Dr. Ray Grubbs and Dr. George M. Caldwell, who is now retired. He served as county medical examiner for 25 years.

Dr. Showalter came to membership in The Medical Society of Virginia through the Southwestern Virginia Medical Society. He belonged also to the Southeastern Surgical Congress, International College of Surgeons, and Society of Medical Consultants to the Armed Forces.

## Paul C. Hendrix, MD

Dr. Paul C. Hendrix, who practiced medicine in Wytheville for 31 years but had recently been living in Winston-Salem, North Carolina, died August 19 in a Winston-Salem hospital. He was 60 years old.

After graduating from Emory University School of Medicine in Atlanta, Dr. Hendrix trained in Winston-Salem, then set up a general practice in Wytheville. He had served as chief of staff of Wythe

County Community Hospital and was a past president of the Wytheville Rotary Club. His membership in the Southwestern Virginia Medical Society and The Medical Society of Virginia spanned almost 40 years.

### **Paul D. Camp, Jr., MD**

Dr. Paul Douglas Camp, Jr., died on January 22 at his home in Richmond. He was 81. Dr. Camp established what was thought to be Richmond's first practice limited to cardiology and founded the outpatient cardiac clinic at the Medical College of Virginia Hospital.

A native of Franklin, Dr. Camp graduated from Virginia Military Institute and the University of Virginia School of Medicine, Class of 1928. He trained at Roosevelt Hospital in New York, then was a Darden fellow and cardiac resident under Dr. Paul Dudley White at Massachusetts General Hospital. He studied abroad in London and Vienna, then returned to Richmond in 1934 to establish his practice, which grew to become Medical Associates of Richmond, with Dr. Camp as chairman. He retired from practice in 1978.

Dr. Camp was on the faculty of the Medical College of Virginia and served as a consultant to the Rheumatic Fever Children's Heart Clinic and the Rheumatic Fever Program of the State Health Department. He had been a governor for Virginia of the American College of Cardiology and president of the Tri-State Medical Society and the Richmond Area and Virginia Heart Associations. His membership in the Richmond Academy of Medicine and The Medical Society of Virginia spanned 51 years and he belonged also to the American College of Physicians.

## **Memoir of R.E. Glendy 1902-1983**

*By Charles L. Crockett, Jr., MD*

Dr. Robert Earle Glendy, who died June 26, 1983, at the age of 81, spent most of his early life in Dublin, Virginia. He was graduated from Virginia Military Institute in 1925 and from the University of Virginia School of Medicine in 1931. He married a classmate, Dr. Margaret Moriarty, who died in 1978. He interned in medicine at the Peter Bent Brigham Hospital in Boston under Dr. Henry Christian, and there his interest in cardiology was first

kindled by an association with Dr. Samuel Levine. He took further residency training at the Massachusetts General Hospital, where he became a Dalton Scholar in medicine and fellow in cardiology under Dr. Paul Dudley White. He was associated in practice with Dr. White and was an instructor at Harvard Medical School from 1936 to 1941. He served stateside and overseas in the Army Medical Corps as a lieutenant colonel from 1941 to 1945. He practiced internal medicine and cardiology in Roanoke from 1945 to 1968, following which he served as director of cardiology at Roanoke Memorial Hospitals until his retirement in 1977. He was a clinical scholar in medicine on the University of Virginia faculty.

He was a diplomat of the American Board of Internal Medicine and a member of the American College of Physicians, American Society of Internal Medicine, American Medical Association, American Heart Association, Roanoke Academy of Medicine, The Medical Society of Virginia, and the Southwestern Virginia Medical Society. He was a past president of the Roanoke Valley Heart Association, Roanoke Academy of Medicine, and Virginia Heart Association, and had served as chief of staff at Roanoke Memorial Hospitals.

Upon entering practice in 1945, Dr. Glendy pioneered the specialty practice of cardiology in Roanoke and the Western Virginia area. He stimulated and encouraged young cardiologists to enter practice in the region. In recognition of his many contributions to cardiology, the Roanoke Valley Heart Association established the Glendy Lecture-ship in 1973 to be given with its annual cardiac symposium, which program is conducted in conjunction with the Roanoke Memorial Hospitals' annual postgraduate program. Dr. Glendy helped to nurture the latter in its early years.

Earle was highly respected by his colleagues as a skilled internist and consulting cardiologist. His patients had total confidence in the careful, conscientious care he always provided them. His friends were legion and his company was highly sought after by those in diverse walks of life who enjoyed him as a hunting companion or for a round of golf. When gathered with a group of friends, there was always the inevitable request for Earle to "tell a story". His unique way with words brightened the lives of his friends, students, and colleagues on many occasions.

In 1970 he received the brotherhood citation award of the National Conference of Christians and Jews. His contributions to the medical community and patients he served and his influence in a much



broader range are immeasurable and will be long remembered.

He is survived by his wife, Stella Barbee Glendy; sons Robert Earle Glendy, Jr., and David Gardner Glendy; daughter Lisa Glendy Hull; stepdaughters Barbara Barbee Bell, Beverly Barbee Belcher; and nine grandchildren.

## Memoir of R. M. Miskimon 1914-1983

*By Alan Hecht, MD, Joseph C. Parker, MD,  
and William R. Jones, Jr., MD*

Dr. Robert Murray Miskimon died August 24, 1983, after having successfully fought with courage and good humor the cardiac disease which had plagued him for the previous six months.

Dr. Miskimon was born in Washington, DC, received his undergraduate education at Dartmouth College and his medical degree from the College of Physicians and Surgeons of Columbia University in New York.

The four years following his graduation were spent in the United States Army Medical Corps during World War II, at which time he saw service as chief of the medical section on the 23rd General Hospital and combat duty during the invasion of Salerno.

He came to Richmond following his military service and practiced internal medicine from 1946 until 1956, during which time he also held a number of industrial medical positions, including the medical directorship of the Richmond City Employees' Medical Service.

In 1956 he gave up his private practice and became medical director of the then newly founded Fidelity Bankers Life Insurance Company, a position he held until his retirement in 1977. He and the company grew together, and his contributions to the company's present success and his concern for the well-being of each of his fellow employees were immeasurable. The thoughtfulness, compassion and skill with which "Dr. Bob", as he was known, faced both employee health and corporate medical problems will long be remembered.

Always a busy and active man, Bob Miskimon was a member of numerous professional societies, including the American Medical Association, The Medical Society of Virginia, Richmond Academy of Medicine, Association of Life Insurance Medical Directors, and the Virginia Industrial Medical Association, of which he was a past director.

His wife, Archer C. Miskimon, and their children shared with him his interest in nature and particularly his love and respect for the sea. He enjoyed boating, and had been a member of the US Coast Guard Auxiliary, a district officer of the US Power Squadron, and a commander of the Richmond Power Squadron. He was also an accomplished amateur photographer and often used his photographs to share with others his interest in the world of nature.

Those who knew Bob Miskimon as a friend will miss him, for he taught us much, and his memory will refresh and sustain us for the rest of our lives.

## Memoir of Fred Delp 1905-1983

*By Julian B. Doss, MD, David A. Garner, MD  
and William D. Poe, MD*

Dr. Fred Delp, age 77 years, died September 30, 1983 at his home in Roanoke, Virginia, following an extended illness.

Fred was a native of Rural Retreat, Virginia, the son of the late Dr. W. Guy Delp and Bessie Bumgardner Delp. He is survived by his wife, Virginia Young Delp; three children, Jeffrey Delp, Holland, Michigan; Ashley Delp Nackley, Piedmont, California, and Deborah Delp, Raleigh, North Carolina; and six grandchildren.

Dr. Delp was a graduate of Washington and Lee University and the Medical College of Virginia, with a doctor of medicine degree, class of 1934. He served internship at the University of Michigan Hospital. He was a family practitioner in Pulaski, Virginia, for 26 years. He then served as staff physician at the Veterans Administration Medical Center in Salem for the next seven years. He retired in 1969 due to failing health.

Dr. Delp was a member of the American Academy of Family Physicians, the Flying Physicians Association, American Medical Association, The Medical Society of Virginia, and the Virginia Academy of Family Physicians, which he served as editor of the medical magazine for several years. He had served as president the Southwest Virginia Medical Society and was a member of the Roanoke Academy of Medicine and of Trinity Lutheran Church, Roanoke.

Dr. Delp presented interest and patience in his medical practice. He was esteemed by his patients and friends, possessed a good sense of humor, was tranquil during his final illness and always presented a ready smile.

# WHO'S WHO

Expert anglers and angiographers have a lot in common, according to **Dr. Charles J. Tegtmeier**, director of angiography at the University of Virginia and world-class tarpon fly-rod fisherman. "Both require a lot of gadgets and hand-eye coordination," he explains.

Dr. Tegtmeier has been hooked on fishing since his boyhood days in upstate New York. His affinity for medicine came early, too, through both his father, who was a surgeon, and his mother, who was a scrub nurse. After a detour into geology at his father's alma mater, Colgate University, "Tunk" Tegtmeier hit the high road to medicine at George Washington University, earning his doctorate in 1965.

Three years of residency in surgery followed, then he switched to diagnostic radiology. Before joining the University of Virginia faculty in 1972, he served a one-year fellowship in cardiovascular radiology at Harvard University's Peter Bent Brigham Hospital.

Today he is professor of radiology and associate professor of anatomy at UVA, and his lifelong passion for outdoor activity has led him into sports medicine as a specialist in knee arthrography.

Early next year Dr. Tegtmeier is to be installed as president of the Society of Cardiovascular and Interventional Radiology, an organization founded in 1974 and dedicated to the radiologic subspecialties of diagnostic and therapeutic angiography and interventional radiology.

Dr. Tegtmeier's peers recognize him as an interventionalist of national reputation for his proficiency

with the balloon catheter procedure known as percutaneous transluminal angioplasty, which is used for treating kidney-related high blood pressure. Other interventional procedures include abscess drainage, embolization, fibrinolysis, and biliary and urinary drainage. And even though the university's new lithotripter is now disintegrating kidney stones, percutaneous removal will continue to be the treatment of choice in some patients.

As facile with a pen as with a catheter, Tunk Tegtmeier is the author of 70 articles in the literature and has written on fishing for sports publications.

His wife, Virginia Peters Tegtmeier, PhD, works at the University of Virginia, too, as a counselor, and obligingly shares his enthusiasm for fishing. They like to fish for bass near their home on the Rivanna River reservoir, and Dr. Tegtmeier competes in national and international fly-fishing tournaments off the Florida coast. He set a Florida state record for 1982 when his fly rod landed a 139½-pound tarpon.

Membership in the Society of Cardiovascular and Interventional Radiology is limited to 100 members. Invitations to join are issued after intensive peer review. Dr. Tegtmeier is to be installed as president at the society's meeting in March 1985 in Orlando, Florida.

**Dr. Leo Goldhammer**, Arlington, is a newly elected member of the American Academy of Neurology.

A member of The Medical Society of Virginia's new Section for

Resident Physicians was among 20 recipients of awards of \$1,500 from the American Academy of Family Physicians. She is **Dr. Susan Marie Pollart**, who was graduated from the University of Virginia School of Medicine in 1982 and is now a resident in family practice.

Another winner among the resident physicians of the new MSV section is **Dr. Richard C. Sadove**, who won first prize in the 1984 scholarship contest of the Educational Foundation of the American Society of Plastic and Reconstructive Surgeons. Dr. Sadove is in the Eastern Virginia Graduate School's department of plastic surgery.

A plaque honoring **Dr. Thomas J. Moran** as the founder of laboratory medicine in Danville was placed in the new laboratory of Danville's Memorial Hospital. The plaque was commissioned by the hospital's board of directors. Dr. Moran went to Danville in 1946 as the first full-time pathologist at Memorial Hospital. He retired from practice last December.

**Dr. John Jay Kreuger**, Virginia Beach, has been appointed to the State Board of Health for a four-year term by Gov. Charles S. Robb. Dr. Kreuger is a pathologist.

Secretary Margaret M. Heckler of the Department of Health and Human Services has appointed **Dr. Grayson B. Miller, Jr.**, Richmond, to the National Committee on Vital and Health Statistics. Dr. Miller directs the Division of Epidemiology of the Virginia Department of Health.

After 32 years as a busy ob-gyn, **Dr. William N. Reingold** of Norfolk has retired to devote his full attention to painting. Dr. Reingold took up painting as a sideline about seven years ago; it quickly became a



lot more interesting than delivering babies.

A Norfolk native, Dr. Reingold graduated from Virginia School of Medicine. Now 58, he is much too young to retire, complain his associates and his patients, but when interviewed by Donna Mason for the Norfolk Virginian-Pilot, Dr. Reingold admitted to being a bit tired after delivering upwards of 9,000 babies and added, "I don't want to be like the athlete who plays one game too many. This profession has taken a great deal of self-discipline. And I want so badly to paint."

When shown at Norfolk's Chesapeake Bay Gallery in July, a collection of Dr. Reingold's pre-retirement paintings brought "a great response," according to the gallery's owner. He works entirely in acrylics. Maritime scenes are recurring subjects, although Williamsburg recently caught his painterly eye, and when he set off in late summer for a trip to Alaska and Canada, he took his paints with him.

In Charlottesville, too, there's an ob-gyn with a passion for painting, but at the age of 37 he isn't about to retire for it. He is **Dr. Scott D. Vogel**, who has been painting since his fourth year at the University of Wisconsin School of Medicine. Oils are Dr. Vogel's medium. Landscapes in the realistic manner are his artistic specialty, and for a subspecialty, the dramatic cloud formations that occur over the foothills of the Blue Ridge Mountains, he has made a study of meteorology. Dr. Vogel recently loaned a group of his works to the University of Virginia Theater for display in the theater's lobby, and a Charlottesville art critic who reviewed the collection praised his work as "flawlessly executed . . . with a grandeur seldom seen in the creations of a self-taught artist."

A native of Wisconsin, Dr. Vogel married the former Lucie Leavell, whose father, the late **Dr. Byrd S. Leavell**, was for many years a professor of medicine at UVa; it was she who led Dr. Vogel to settle in Virginia.

**Dr. Joseph T. McFadden** of Norfolk works in oils in his off hours, too, but not the kind you put on a palette. He's into machine oil, to keep an industrial lathe and a one-ton milling machine humming in the workshop of his home. There he designs and crafts medical tools that make brain surgery easier for him and safer for his patients.

Chairman of the Department of

Neurosurgery at Eastern Virginia Medical School, Dr. McFadden apprenticed to two veteran machinists to learn their craft. He has also become something of a metallurgist, with a library of 2,500 books on metals. For a headrest of his design that be adjusted to any angle, he has a patent pending, and his "McFadden clip" is used in operating rooms worldwide for cutting off the blood flow to aneurysms.

Interviewed in his workshop by Ellen Whitford for the Virginian-Pilot, Dr. McFadden observed, "I don't play golf, I sold my boats, so this is what I do. The puzzle of it absorbs you."



**Dr. Tegtmeier inserting balloon catheter. At left, a technician.**

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## A Salute to President Kuykendall

**I**T is with great pride in The Medical Society of Virginia that those who know Harry C. Kuykendall see him assume our leadership.

In a time when we are becoming more and more specialized, Harry brings to the Society's leadership an extraordinary scope of endeavors—an avid swimmer who qualified for the Olympic trials in his youth, a sailor experienced in both cruising and racing, a marksman and a skier. He also is a person well versed in the arts—a professional pianist, a painter, woodcutter, cabinetmaker, gardener and artisan in stained glass.

A direct male descendant of the Dutch colonists who settled in America prior to 1645, Harry is a native Virginian. Brought into this world in Harrisonburg by his uncle, the distinguished Noland M. Canter, MD, Harry grew up in Alexandria and went on to attend college at Randolph-Macon. After serving in Japan as an Army combat medical corpsman, Harry studied biology at the University of Michigan and Florida Southern University. After obtaining his MD degree from the Medical College of Virginia, where he also interned in medicine and surgery, he was a surgical resident at Norfolk General Hospital and a preceptor in medicine and physician-in-charge at Sheltering Arms Hospital, Richmond. In 1966 he returned to Alexandria, where he has remained in private practice.

Harry has devoted his time to the cause of organized medicine generously. On the state level, he not only has served as our President Elect during the past year and as First and Second Vice President, but also as a delegate and councilor from the





8th District. He has served on many state-level committees and as chairman of the PSRO Liaison Committee as well as the Vanguard Committee and 1971 Scientific Session. On the local level, he was president of the Alexandria Medical Society, and a recipient of that Society's seldom bestowed Community Service Award.

In addition to service to his local society and The Medical Society of Virginia, Harry has been active with his specialty group, serving as president of the Northern Virginia Academy of Family Physicians and delegate and director of the Virginia Academy of Family Physicians. He is a charter fellow of the American Academy of Family Physicians and board certified by the American Board of Family Practice.

He was the first president of the Northern Virginia Foundation for Medical Care and also served as its medical director. He is a past president of the Alexandria Chapter of the American Cancer Society, and vice chairman of the Alexandria Board of Health and on the Alexandria Mayor's Committees on Health Care and Peer Review. Currently he serves as a director of the Virginia Professional Standards Review Foundation (since 1974) and has been a governor of the Northern Virginia Consortium for Continuing Education since 1975.

HARRY C. KUYKENDALL, MD,  
PRESIDENT, 1984-1985, THE MEDICAL SOCIETY OF VIRGINIA

Harry's wife, the former Holly Ward, is a past president of the auxiliaries of the Alexandria Medical Society and the Virginia Academy of Family Physicians. They reside in Alexandria, devotedly caring for their obviously brilliant son, Ward Van Halstead, who is now 18 months old.

It is with full confidence in his ability, dedication and clear thinking that the members of The Medical Society of Virginia welcome Harry's accession to the presidency. He is prepared for the post and will do well in responding to the growing pressures of the office.

Ira J. Green, MD

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# Fertility Rite

**D**rawn by the renown of the in vitro fertilization program at Eastern Virginia Medical School, specialists from all over the world converged on Norfolk in late October for the Third Annual Symposium on Human Reproduction. For five days the visiting physicians listened to reports from the fast-changing fields of reproductive endocrinology and extracorporeal fertilization with embryo transfer. None of the speakers was more closely attended than Dr. Howard W. Jones, Jr., and Dr. Georgeanna Seegar Jones, the husband and wife who in 1978 transferred themselves from the Johns Hopkins School of Medicine to develop at EVMS the nation's seminal in vitro program. The first baby born of that program will be 3 years old this month, and across the country there are now 58 similar programs.

Sponsoring the symposium were the Howard and Georgeanna Jones

Photographs by Larry Branham



**His fluent Spanish landed Dr. Anibal A. Acosta, the symposium's course director, in the translator's booth.**

Institute of Reproductive Medicine and the Department of Biology of Old Dominion University. Created by Eastern Virginia Medical School last year with the Doctors Jones as president and vice president, the Institute is a division of the Department of Obstetrics and Gynecology, of which Dr. Mason C. Andrews is chairman.

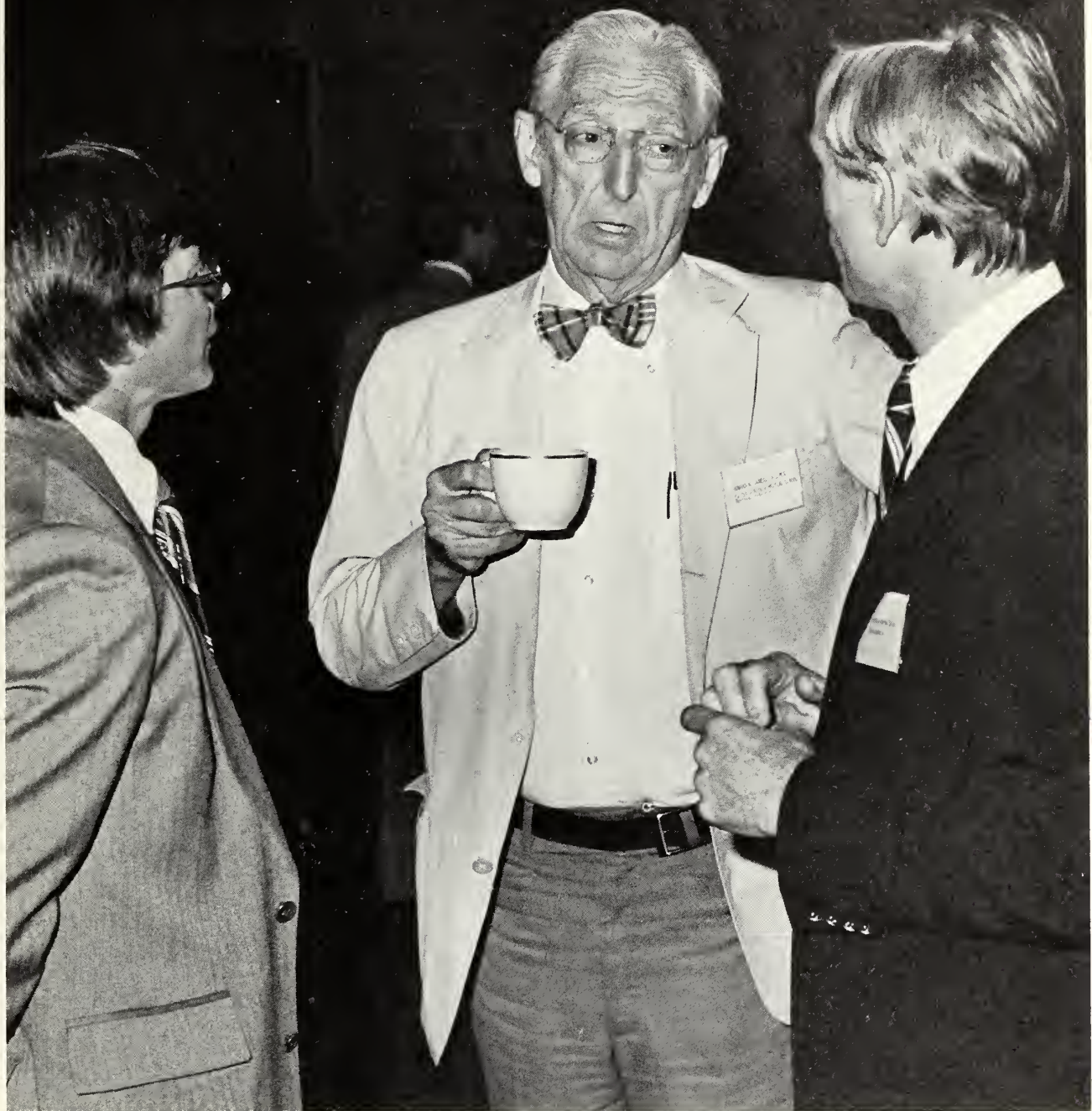
**On the podium, Dr. Zev Rosenwaks, director of the Institute named for the Joneses, makes a point.**



**In the audience, Dr. Georgeanna Seegar Jones listens attentively.**



**Dr. Howard W. Jones, Jr.,  
fields questions during a coffee  
break.**



# MEETINGS

## January 22

**38th Annual Stoneburner Lecture: Rehabilitation Medicine** (Medical College of Virginia), *Richmond*. Kathy Martin, Office of CME, (804) 786-0494.

## January 25-26

**4th Annual Thornton Obstetrics and Gynecology Symposium** (University of Virginia School of Medicine), *Charlottesville*. Office of CME, (804) 924-5318.

## January 28-31

**Alton D. Brashear Postgraduate Course in Head and Neck Anatomy** (Medical College of Virginia), *Richmond*. Hugo R. Seibel, PhD, Box 709, MCV Station, Richmond VA 23298.

## January 24-26

**Annual Meeting of the Neurosurgical Society of the Virginias**, *Hot Springs*. Jacques E. Botton, MD, 1933 Thomson Drive, Lynchburg VA 24501.

## February 4-6

**Winter Retreat: Pulmonary Medicine** (Medical College of Virginia), *Wintergreen*. Fee: \$250. Beth Winn, Office of CME, (804) 786-0494.

## February 14-16

**Pearls and Pitfalls in Emergency Medicine** (Virginia Chapter, American College Emergency Physicians), *Wintergreen*. 16 Cat. 1 hrs. Fee: \$175-\$300. Gwen E. Messler, (804) 737-9433.

## February 20-22

**Thromboembolic and Atherosclerotic Disease—Update on Pathogenesis, Prevention and Treatment** (American College of Cardiology), *Bethesda, Maryland*. 17 ½ Cat. 1 hrs. Learning Center Registrar, (301) 897-5400, ext. 241.

## February 22-24

**Regional Scientific Meeting, American College of Physicians**, *Norfolk*. Dr. James P. Baker, Eastern Virginia Medical School, 600 Gresham Drive, Norfolk VA 23507, (804) 628-3584.

## February 25-27

**Cardiovascular Surgery, Coronary and Valvular** (American College of Cardiology), *Bethesda, Maryland*. Learning Center Registrar, (301) 897-5400, ext. 241.

## February 25-March 1

**Current Concepts in Ophthalmology** (Johns Hopkins Wilmer Institute), *Dorado Beach, Puerto Rico*. Office of CME, Program Coordinator, (301) 955-6046.

## March 4-7

**Annual Postgraduate Course in Radiology: Interventional Techniques and Radiologic Management of the Emergency Patient** (Medical College of Virginia), *Richmond*. Fee: \$425. Kathy Martin, Office of CME, (804) 786-0494.

## March 16

**57th Annual Stuart McGuire Lecture: Reconstructive Plastic Surgery** (Medical College of Virginia), *Richmond*. Beth Winn, Office of CME, (804) 786-0494.

## March 21-22

**Post-Graduate Days** (Roanoke Memorial Hospitals), *Roanoke*. 10 Cat. 1 hrs. Nancy Howell Agee, PO Box 13367, Roanoke VA 24033, (703) 981-7009.

## March 25-30

**Pediatric Trends** (Johns Hopkins School of Medicine), *Baltimore*. 45 hrs. Fee: \$500. Justean Coles, Office of CME, (301) 955-5880.

## March 29-31

**Ophthalmology: Cataract Surgery and Intraocular Lenses**, (Medical College of Virginia), *Williamsburg*. Fee: \$275. Kay Parrott, Office of CME, (804) 786-0494.

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# WHO'S WHO

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## IN VIRGINIA MEDICINE

"Dr. Bernart is retreating just a bit," read the headline in the Eastern Shore News. The reference was to **Dr. William F. Bernart** of Nassawadox. He is retreating, the story under the headline explained, from a work schedule of 80 hours a week to "a mere 30 to 35 hours."

Specifically, Dr. Bernart is retiring from his activities at Northampton-Accomack Memorial Hospital in Nassawadox, and thus, as reporter Jack Davis put it, "the second shoe is dropping." The first shoe fell two and a half years ago when **Dr. William Stewart Burton** had to give up his practice at the hospital because his heart was acting up. Dr. Burton and Dr. Bernart had been mainstays at the hospital since the late fifties.

"For 12 years," the story recounted, "the two covered internal medical services at the hospital 24 hours a day; they alternated the night duties, which included the emergency room, each relieving the other. And that also involved treating up to 250 migrant laborers during the summer." (They finally got some help in 1971, when the hospital began hiring Norfolk doctors to moonlight in the emergency room. Today the ER is managed by a group of doctors under contract.)

At 57, Dr. Bernart observed to the reporter, he's looking forward to slowing down. He'll schedule office hours four mornings and three afternoons a week, and he'll continue as medical director of Heritage Hall, a 120-bed nursing

home, and as volunteer physician with the Delmarva Rural Ministries in the medical clinic for migrants, but he'll spend more time on the water, fishing and sailing, which is what brought him to the Eastern Shore in 1958.

Born in Providence, Rhode Island, and reared in Connecticut, Bill Bernart joined the Navy during World War II, then went to Princeton and the Columbia University School of Medicine. It was at Columbia that he met a pretty nurse on the psychiatric service; her name was Cindy, and she became his wife.

Dr. Bernart's training took them first to Harvard, then to the University of Puerto Rico (a National Heart Institute traineeship), and then to the University of Virginia. Scouting Virginia's waterways for a place to live and practice, the Bernarts found Nassawadox, and there they have been ever since, rearing four sons and a daughter, all now grown.

The Bernarts have been instrumental in luring other physicians to Nassawadox, making recruiting visits to medical schools at their own expense, then housing and entertaining prospects who came to the Shore to look things over. Among those who stayed are **Dr. Henry B. Dixon II**, whose specialty is cardiovascular diseases; **Dr. William W. McIntyre**, gastroenterologist; **Dr. James L. McDaniel**, pediatricist; and **Dr. Harry S. Holcomb III**, orthopedic surgeon.

Another big chunk of Dr. Bernart's time and attention has gone into the hands-on training program for medical students begun in 1967 by the Medical College of Virginia. Northampton-Accomack Memorial Hospital was one of four hospitals designated for this program, and under the joint direction of Bill Bernart and Bill Burton, the program has flourished there.

The Bernart-Burton team has

made notable contributions to organized medicine, too. Both have served as president of the Northampton County Medical Society. Dr. Bernart also was for many years a delegate to The Medical Society of Virginia's annual meetings, while Dr. Burton is now MSV councilor from the 1st district.

**Dr. John William Selman**, Martinsville, has been elected a fellow of the American Academy of Facial Plastic and Reconstructive Surgery.

A teaching center to be built in the University of Virginia's new hospital is to bear the name of the late **Dr. Paul D. Camp, Jr.**, retired Richmond cardiologist who graduated in 1928 from the university's School of Medicine. Dr. Camp's sister, Mrs. Charles R. Younts of Atlanta, the former Willie Camp, and her husband have pledged \$1 million toward the center, which is to be a conference facility or auditorium for clinical meetings, classes and similar activities devoted to cardiology.

Two Virginia Beach physicians, **Dr. Rolfe D. White** and **Dr. David L. Williams**, are the authors of a chapter in the new second edition of *Ultrasound in Urology*, published by Williams & Wilkins, Baltimore. The title of their contribution: "Dynamic Evaluation of the Lower Urinary Tract." The book was edited by Martin I. Resnick and Roger C. Anders.

**Dr. Shane J. Kraus**, whose general practice is centered in Providence Forge, toured Australia and New Zealand in October as one of a 28-member delegation representing public health and preventive medicine in Virginia under the auspices of the People-to-People program.

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# Feast or Famine

In our society, eating habits vary, and food is often used as more than a source of nourishment. So we accept many individualized patterns.

But some people go too far. The anorectic, threatened by food, slips into a pattern of self-imposed starvation. The bulimic, using food as a narcotic, binges and purges in an attempt to cope with overpowering emotions.

The problems demand expert intervention of an unusually comprehensive sort. They demand nothing short of what Sheppard Pratt now provides in its Eating Disorders Program.

Just as the ideal diet for any individual is a matter of proper balance, the ideal treatment for anorexia or bulimia must also be properly balanced and individualized. For this reason, Sheppard Pratt carefully evaluates each patient and tailors a treatment program that draws on all necessary disciplines: medicine, psychiatry and social work. And we administer this treatment intensively, continuously—from inpatient to outpatient to aftercare.

Sheppard Pratt is achieving good results with eating-disordered patients by countering the extremes of feast or famine with a steady diet of multi-disciplinary guidance and care.

For a more detailed description of the Eating Disorders Program at Sheppard Pratt, please contact: Dr. David Waltos, Admissions Officer, Sheppard and Enoch Pratt Hospital, P.O. Box 6815, Baltimore, MD 21204. (301) 823-8200.



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# LETTERS

## Remembers Dr. Haller's fine tribute to teachers

Those of us who were there remember the stimulating lecture entitled "The Importance of Preceptors in Medical Education—Hitch Your Wagon to a Star" of Dr. J. Alex Haller, Jr., MD, presented as the 1984 Walter Reed Lecture before the Richmond Academy of Medicine Historical Section. Dr. Haller was born in Pulaski, Virginia, the son of the late Dr. J. Alex Haller, Sr., of Wytheville and Pulaski, Virginia.

Dr. Haller's lecture was primarily devoted to paying tribute to others for their dedicated scientific investigation. In his historical survey he mentioned the various years of investigations, showing the successful response to correcting congenital anomalies, including tetralogy of Fallot, a heart malformation frequently called "blue baby."



J. Alex Haller, MD

Dr. Haller gave the background of many centuries in surgery, even paying tribute to the "barber surgeons," showing that some of their methods worked better than those practiced by university graduates in internal medicine in the medical world at that time. He recognized the work of dedicated technicians, particularly Mr. Vivien Thomas at Johns Hopkins, who with others had worked with Dr. Alfred Blalock to develop the final successful techniques enabling cardiac surgeons of today to correct the heart anomalies causing "blue baby."

Feeling grateful for his experience as a preceptor to these great teachers and the help of others, Dr. Haller wished to remember those that had helped him, particularly Mr. Thomas, and requested the Johns Hopkins University board of trustees to give special recognition to Mr. Thomas, maybe an honorary BS degree. But the trustees, realizing the significant contribution made by this team and Mr. Thomas' proven techniques, awarded him instead an honorary Doctor of Medicine degree, a step rarely taken by any medical school!

It was a great pleasure to hear Dr. Haller's dynamic presentation with accompanying photographs and slides, given so fluently to a very attentive audience.

L. Benjamin Sheppard, MD

116 East Franklin Street  
Richmond VA 23220

## Fees instituted for handling specimen collection kits

As a result of the recent Critical Reevaluation of State Government, the General Assembly approved a change in the Code of Virginia permitting the Department of General Services to charge a handling fee for certain specimen collection kits submitted by the medical professions to our microbiology laboratories. A reduction in our operating budget for 1984-86 was also approved by the General Assembly to reflect the anticipated income from these fees.

We now require that physicians, clinics, hospitals, and other medical facilities in the private

sector purchase stamps for certain laboratory specimen kits. Below is a list of these kits and charges.

As proof of payment, these stamps must be affixed to the back of the specimen history/report form when the specimen is submitted for examination. Specimens submitted in laboratory collection kits from other sources will not be accepted. Specimens received without stamps will be tested, but a bill for handling will be sent with the laboratory report.

Public health facilities (state and local) are exempted from these charges. They will be expected to write the word "exempt" on the top of the history form when they submit fee-requiring kits.

Order forms for requesting stamps for laboratory specimen kits may be obtained by calling or writing to the following office: Department of General Services, Division of Consolidated Laboratory Services, Attn: Mrs. Jane Beagle, 1 North 14th Street, Room 231, Richmond, VA 23219, (804) 786-3274. Money orders or checks must be made out to the Treasurer of Virginia. The specimen kits themselves may be ordered as previously, from the Microbiology Laboratory in Richmond, Luray, or Abingdon.

Charging handling fees is a new venture for the laboratory, so we solicit your cooperation and understanding.

F. W. Lambert, DrPh

Bureau Director, Microbiological Science  
Department of General Services  
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#### SPECIMEN KIT HANDLING FEES

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## Women, children need WIC support, WIC needs physician support

In the ten years since its congressional authorization, the Special Supplemental Food Program for Women, Infants and Children, more popularly known as WIC, has expanded nationwide and now serves approximately 3 million participants through WIC's 7,000 clinic sites.

The WIC Program is unique in that it provides individually tailored food packages and nutrition education to pregnant, postpartum and breastfeeding women, infants and children up to age 5 who are determined to be at nutritional risk based upon medical assessment. Infants receive iron-fortified formula and when 4 months old, iron-fortified cereal and juice high in Vitamin C. Women and children receive milk, cheese, eggs, beans or peanut butter, iron-fortified cereal and fruit juice. Participants use WIC checks to purchase these foods at any WIC-authorized grocery store, pharmacy or military commissary in the State.

Research on the benefits of the WIC Program has revealed some impressive facts. Low-income women are seeking prenatal care at significantly earlier stages of pregnancy and the practice of breastfeeding is once again on the rise thanks to more extensive education on the subject. Data from several large-scale studies show that women who participate in WIC deliver fewer low-birth-weight infants, and a Harvard School of Public Health Study found that every dollar spent on the prenatal component of WIC results in a savings of \$3 in hospitalization costs, due to the reduced number of low-birth-weight infants needing extended hospital care.

The Virginia WIC Program operates from 153 clinic sites across the state and currently serves a monthly caseload of nearly 64,000 participants. Despite this figure, it is estimated that 92,000 other Virginians who are eligible for WIC assistance are missing out, and the Virginia WIC Program has intensified its outreach efforts in an attempt to make the public more aware of this service.

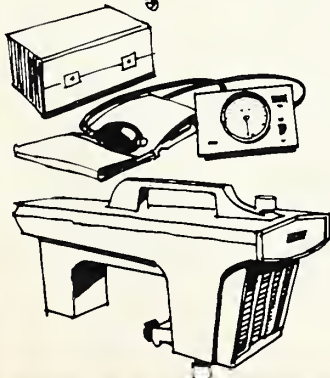
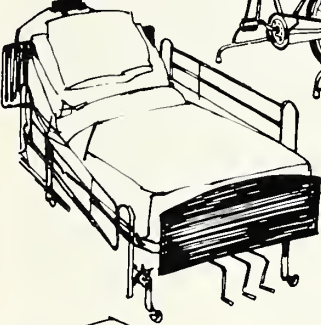
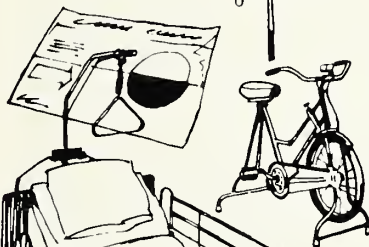
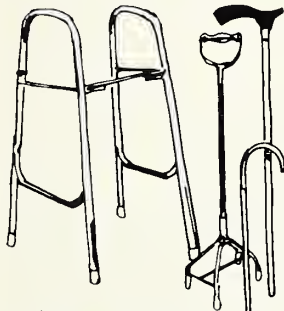
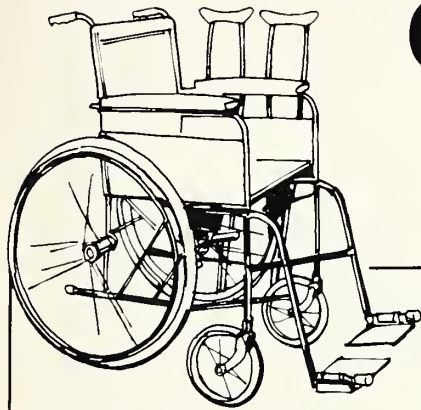
It is imperative that Virginia's physicians support the program through patient referrals and a general knowledge of WIC's benefits and operational procedures. Private physicians may obtain more information and WIC Program referral forms at their local health department or at the WIC office at the address below.

Brenda Morgan

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## PEOPLES DRUG

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# Chest Pain with Pulmonary Infiltrate: Clinicopathology Conference

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From the Department of Medicine,  
University of Virginia School of Medicine  
Discussed by Lockhart B. McGuire, MD

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## PRESENTATION OF CASE

DR. JOHN SCHORLING: A 56-year-old woman was admitted to the medical intensive care unit at the University of Virginia Hospital with chest pain and a pulmonary infiltrate and died 24 hours later.

The patient was a housewife who had been in excellent health until the morning of admission. She awoke that day feeling well but, while preparing breakfast, suddenly developed severe substernal pain, shortness of breath accompanied by a smothering sensation, and nausea without vomiting. She described her chest pain as being both a sensation of constant, heavy pressure and a sharp, stabbing pain on deep inspiration. The pain did not radiate, but she did complain of numbness in her left little finger. She experienced no associated light-headedness, diaphoresis or palpitations. Shortly after the onset of pain, she coughed up a teaspoonful of blood-streaked sputum. She denied having chronic

cough, increased sputum production, fever, chills, malaise, myalgias or headache.

The patient had no history of chest pain, heart disease, or symptoms of congestive heart failure. She had no history of deep venous thrombosis, immobility, recent surgery, estrogen use, or pain and/or swelling in the calf. Her past medical history was remarkable only for a ten-year history of mild hypertension; this had not been treated for three years because her blood pressure had been within normal limits. A hysterectomy was done 25 years previously. She had smoked one pack of cigarettes per day for the last 35 years. She was taking no medications. She had once developed hives after ingesting penicillin. Her father, one sister and one brother had died in their 50s of myocardial infarctions. There was no personal or family history of diabetes mellitus.

On physical examination, the patient was a slightly obese white female complaining of severe substernal pain which waxed and waned spontaneously. Blood pressure was 80/50 mm Hg in both arms supine, pulse 74/min and regular, respirations 16/min and rectal temperature 37°C. The skin was cool and dry. There was no adenopathy. HEENT examination revealed only slight bilateral arteriolar nar-

Presented on 9-7-83. Edited by John Schorling, MD, Erik Hewlett, MD, and Marcia Day Finney. Address correspondence to Mrs. Finney at Box 466, Department of Medicine, University of Virginia School of Medicine, Charlottesville VA 22908.



rowing. The neck was supple and without thyromegaly. Chest examination showed no evidence of trauma and no tenderness. While the left lung was clear, there were rales in the lower right posterior lung field. There were no rubs. Cardiovascular examination revealed no jugular venous distention. The cardiac apex impulse was normal.  $S_1$  and  $S_2$  were normal and there was an  $S_3$  gallop but no  $S_4$ , rub or murmur. Pulses were 2+ and equal throughout, with no bruits. Abdominal examination was benign and showed no organomegaly. Examination of the extremities showed no clubbing, cyanosis, edema or cords, and Homan's sign for venous thrombosis in the calf was negative. Rectal examination was deferred; the stool was guaiac-negative. Neurological examination was normal.

Admission laboratory data included hematocrit of 36.5%, hemoglobin 12.9 g/dl, mean corpuscular volume 88.8 fl, mean corpuscular hemoglobin 31.5 pg. White blood count was 16,300/mm<sup>3</sup> (10 bands, 76 segs, 12 lymphocytes, 1 monocyte, 1 basophil) and platelets were 206,000; prothrombin time was 11.7/11.9 sec and partial thromboplastin time was 27.2 sec. SMAC was within normal limits. LDH was 170 IU and creatine phosphokinase was 51 IU. Arterial blood gases showed pH of 7.44,  $pCO_2$  27, and  $pO_2$  77 on nasal oxygen, 2 L/min. The chest x-ray was an AP sitting film which revealed moderate cardiomegaly, a right lower lobe lung infiltrate and a questionable right upper lobe infiltrate. The electrocardiogram showed a normal sinus rhythm at a rate of 80/min, with prolongation of the QT interval (QTc 0.47 sec) and mild nonspecific ST-T wave abnormalities. Sputum gram stain showed many red blood cells, occasional white blood cells, and moderate amounts of mixed flora with gram-positive diplococci predominating.

**Hospital course:** The patient's blood pressure increased to 118/60 mm Hg following intravenous infusion of 2 liters of normal saline solution. She was given 8,000 units of intravenous heparin and a continuous heparin infusion was begun, as was intravenous erythromycin, 500 mg every 6 hours. The patient continued to complain of paroxysms of chest pain aggravated by inspiration and only partially relieved by morphine sulfate. Despite a rise in blood pressure to 120/80 mm Hg, she remained oliguric, and, therefore, a Swan-Ganz catheter was inserted. Initial hemodynamic parameters included a pulmonary artery pressure of 30/15 mm Hg, pulmonary capillary wedge pressure of 15 mm Hg and a central venous pressure of 14 mm Hg; cardiac output was 4.5 L/min. Her urine output increased from 10 to 70 ml/hr after 70 mg of intravenous



**Fig. 1:** Sitting AP chest radiograph shows cardiomegaly and extensive perihilar infiltration in right lung.



**Fig. 2:** Left lateral chest radiograph demonstrates obliteration of right hilum, prominent aortic arch, infiltrate extending posteriorly.

furosemide. Her temperature peaked overnight at 38.6°C (rectal).

On the morning after admission a ventilation/perfusion lung scan was attempted, but the patient suffered a cardiorespiratory arrest during the ventilation portion of the study. Cardiopulmonary resuscitation was begun and she was taken to the operating room, where she died during an emergency thoracotomy.

### RADIOLOGY

DR. PAUL DEE: The first x-ray of this patient is a sitting anteroposterior film (Fig. 1), but even allowing for that, the heart is obviously enlarged. In the right lung there is infiltration extending out from the hilar regions with peripheral sparing. This is what we loosely term a "pulmonary edema" pattern. On the left side, there is no corresponding infiltration. There is no evidence of peripheral pulmonary edema—no Kerley's lines, no fluid in the angles—so the best we can say is that there is an extensive central perihilar infiltration in the right lung. The aortic arch looks quite normal in this film.

In the lateral projection (Fig. 2), the normal anatomy of the right hilum is completely obliterated. The right pulmonary artery is not visible end on. The infiltration extends into the lower part of the right oblique fissure of the lung and into the anterior segment of the right lower lobe. The ascending aorta appears somewhat prominent; I cannot quite delineate it from this angle, but it comes very close to the back of the sternum. There is thus some suggestion of aortic dilatation, but this is not particularly surprising given the patient's long history of hypertension.

### CLINICAL DISCUSSION

DR. LOCKHART B. MCGUIRE: When I first read this protocol, I thought, "How nice, they're asking me to discuss a case of pulmonary embolism." However, as I read and reread the protocol, the likelihood of this being a case of pulmonary embolism diminished, for I found more and more features that could not be explained by a pulmonary embolism or, in fact, by any single clinical entity.

I am troubled by the finding on x-ray of left ventricular enlargement. If this patient's hypertension was indeed so mild that antihypertensive drugs were discontinued three years before she developed this illness, I am left without a good explanation for the left ventricular enlargement. Also, her electrocardiogram does not suggest a long-standing pressure overload on the left ventricle. I may have to

reach my conclusions about this case in the absence of a satisfactory explanation for the left ventricular hypertrophy beyond a guess that more significant hypertension was in fact present than was suggested by recent blood pressure readings.

There are four possible explanations to consider for this illness, which began with severe chest pain and ended in death about 24 hours later. One possibility is acute myocardial infarction, in which case the initial abnormal chest x-ray would have represented the early onset of pulmonary edema. This patient's normal presenting electrocardiogram is not incompatible with a diagnosis of acute myocardial infarction; it is quite possible for an electrocardiogram to be normal within the first hour, or even the first four hours, of an eventually fatal acute myocardial infarction. However, I am going to reject myocardial infarction as a diagnosis.

My decision is based principally on consideration of the pain the patient experienced, which she described as both a sensation of weight and a sharp, stabbing pain aggravated by inspiration. Such pain is quite incompatible with acute myocardial infarction and suggests instead a thoracic, skeletal, pleural or pericardial focus. Were we able to question this patient again, it would be useful to know whether the pain was at its maximum intensity at onset; the description we do have—that of severe substernal pain, waxing and waning spontaneously—seems to indicate that it was abruptly intense rather than of gradual intensification. The pain associated with acute myocardial infarction, pulmonary embolism or pericarditis is never characterized in this way but that reported with dissecting aneurysm of the aorta often is. A final argument against a diagnosis of myocardial infarction in this case is based on hemodynamic data obtained during the patient's brief hospital stay. The measurements of the pulmonary capillary pressure (an indirect measurement of left ventricular diastolic pressure), right ventricular diastolic pressure and central venous pressure are all slightly elevated, normal on the left side being about 10 mm Hg and normal on the right being about 5 mm Hg. Myocardial infarction is predominately a left ventricular event; if this were an acute myocardial infarction leading to death, the left ventricle would be failing out of all proportion to the right ventricle, and the measurements we have do not indicate this. Instead, the remarkable feature of the hemodynamic measurements in this patient is that both sides of the heart seem to be affected equally.

Having set aside the diagnosis of acute myocardial infarction, I would like to look carefully at three



other possibilities—pulmonary embolism, an infiltrative infectious or neoplastic process in the lung that invaded the pericardium and caused pericardial tamponade, and acute aortic dissection.

Pulmonary embolism is a notoriously difficult diagnosis to make. Its signs and symptoms are so varied and nonspecific and only a very few of these findings are present in any given case. It is helpful to group the signs in general categories. A) A number of signs pertain to the *source of the embolus*—for instance, leg vein disease, prolonged immobilization, congestive heart failure or malignancy. B) Of the signs of *pulmonary artery obstruction*, including hypotension, fainting, breathlessness, tachycardia, increased pulmonic closure sound and elevated jugular venous pressure, hypoxia and hypocapnia are the most important; these, however, are overt only when the amount of pulmonary artery obstruction by emboli is equivalent to about half of the cross-sectional area of the pulmonary artery bed—it takes a lot of pulmonary embolic material to produce these findings. C) The signs related to *pulmonary infarction* can be helpful, but they are seldom present. Studies using pulmonary angiograms show that infarction of lung follows only about 10% of emboli.

This patient did have low blood pressure and did have hypoxia with an arterial  $pO_2$  of 77 on two liters of nasal oxygen; she also had a low arterial  $pCO_2$ , and this finding, together with her hypotension, makes pulmonary embolism seem a real possibility. In addition, she had coughed up a scant amount of blood, her chest x-ray revealed a density in one lung, and some of her pain seemed to have a pleural or pericardial quality. It is not difficult to see in retrospect, then, why the physicians caring for this woman, finding some evidence in two categories for pulmonary embolism, initiated heparin anticoagulation at the time they began additional diagnostic studies. I believe that starting anticoagulation treatment on partial evidence is, in general, good policy.

There are several features of this case, however, which should have warned the physicians that pulmonary embolism was not this patient's problem. She experienced no increase in pulse or respiratory rate. There was no complaint of breathlessness—this in a patient who, with a blood pressure of 80/50 mm Hg, would have had significant pulmonary obstruction were embolism the problem. If pulmonary embolism were the principal pathology involved, pulmonary hypertension and systemic hypotension would have been present together. Also, the hypoxemia in this patient was relatively mild for a perhaps devastating amount of pulmonary embolism.

The third piece of evidence against pulmonary embolism—evidence available to me but not to the physicians taking care of the patient—is the fact that she died despite 24 hours of appropriate treatment.

In a 1975 review of pulmonary embolism, Dalen and Alpert<sup>1</sup> estimated that more than 500,000 cases of pulmonary embolism occur annually in the United States. Approximately 10% of the victims die within the first hour of symptoms. If a person survives more than an hour and receives treatment, there is a good chance that the correct diagnosis will be made and treatment will be successful. Of those who survive over an hour and receive prompt anticoagulation, more than 90% survive.

The next possibility I considered was pericarditis of either an infectious or neoplastic cause progressing rapidly to pericardial tamponade, perhaps with intrapericardial hemorrhage as a terminal event. The patient had the pulmonary infiltrate in which could be hidden either infection or a neoplasm, and I notice that she was started on a broad-spectrum antibiotic when anticoagulation was begun. The pain she described was certainly compatible with pericarditis. However, if this were pericarditis, there should have been a friction rub audible early in the illness. At that time, there was not enough pericardial fluid to obscure a friction rub—we know this because the left ventricular contour was still prominent on x-ray. The x-ray on admission was consistent with pericarditis in that chest films in patients with this condition can be normal or can show antecedent myocardial or cardiac disease. The electrocardiogram in pericarditis should be abnormal in some respect, and in this patient it showed none of the abnormalities associated with pericarditis. In early pericarditis, there tends to be widespread ST abnormalities, not only in the horizontal plane leads but in the frontal plane leads as well—a so-called “global” abnormality reflecting the diffuse nature of the pathology throughout the pericardium. Later in the course of acute pericarditis the changes can be nonspecific, but certainly they are present and discernible. This patient's electrocardiogram was entirely normal, except for an interesting prolongation of the Q-T interval.

If, in spite of those mixed features, one hypothesizes that the patient went on to pericardial tamponade, there should have been an increased jugular venous pressure, falling cardiac output and falling blood pressure, all occurring more or less simultaneously. This patient's venous pressure was not initially described as elevated; in fact, it was specifically noted to be not elevated on admission. Never-



**Fig. 3:** Gross photograph of aorta showing dissection of the media.

theless, by the time the pulmonary arterial catheter was inserted, there was symmetrical elevation of central filling pressures, a finding characteristic of tamponade. We do not know whether, in the last few hours of her life, this patient experienced an inspiratory drop in blood pressure and the rise in venous pressure associated with pericardial tamponade. It is quite possible that this patient did die with—and perhaps as a result of—pericardial tamponade, but the evidence that an infection or neoplasm caused it is mostly negative, except for the unexplained pulmonary infiltrate.

The fourth possibility I considered is aortic dissection. Patients who present with acute aortic dissection usually have a history which includes hypertension, although a few patients will have the ocular and skeletal stigmata of connective tissue disease and some will be normal. If I take as evidence the left ventricular enlargement, I can say that this patient probably did have more hypertension than has previously been appreciated. Her

family history is certainly striking for supposed coronary artery disease, and I suspect that she had coronary artery stenosis even though I do not believe that myocardial infarction was her terminal event.

About 85% of persons with aortic dissection have chest pain. This pain is frequently of a mixed character, having an abrupt onset at peak intensity and perhaps some association with motion, position or respiration. We tend to rely too much on the development of either new-onset aortic regurgitation or signs of occlusion of a branch of the aortic arch to indicate that aortic dissection is occurring. Various series on acute aortic dissection report that signs of new-onset aortic regurgitation or new-onset occlusion of an artery in the aortic arch were found, when looked for, in only 30%-40% of persons with documented aortic dissection. Given these handicaps, what about this patient's chest x-ray? The widened aorta which Dr. Dee was looking for would be a helpful clue, but it is by no means seen in all cases of dissection.

Having considered all four possibilities, I believe that this patient presented with incomplete rupture or dissection of a thoracic aortic aneurysm, with some bleeding into the right lung, which accounted for the admission chest x-ray findings.

## PATHOLOGY

**DR. BENJAMIN C. STURGILL:** We did this autopsy after the patient had been explored in the operating room. At the time of autopsy, there were bilateral hemothoraces; we found 400 cc of blood on the left and 450 cc of blood on the right. There was an extravasation of blood in the epicardium at the root of the aorta and around the adventitia of the proximal aorta. More to the point, there was a dissection in the proximal aorta, confirming Dr. McGuire's diagnosis.

This dissection was a proximal or type A dissection which involved the entire length of the thoracic aorta. At autopsy, the dissection (Fig. 3) was largely empty of blood, but one could see a film of blood and a clear-cut splitting of the media between the inner two-thirds and outer one-third of the wall. We found no blood in the pericardial space at autopsy, but it may be that blood was evacuated at the time of surgery.

Left ventricular hypertrophy was documented at autopsy. The heart weighed 450 grams, fully 100 grams above the upper limit of normal for women. In the absence of other explanations for this cardiomegaly, such as valvular disease or primary myo-



cardial disease, we would attribute it to hypertension.

When we studied a portion of the dissection microscopically, we were able to see the split in the media of the aorta where the dissection occurred and cystic medial necrosis or chromotropic degeneration, a condition of the aortic media which predisposes to dissection. Erdheim first called attention to this abnormality in patients with aortic dissection. If this kind of dissection is to develop, the media of the aorta must first undergo certain degenerative changes.

The mechanism of dissection itself is somewhat controversial. Some investigators suggest that it results from a tear in the intima and a forcing of the arterial blood under arterial pressure through this tear and into the defective media. An intimal tear was found in this particular case, but it had been included in the aortotomy incision and sewn together. But not all aortic dissections will show such an intimal tear. This has suggested to other investigators that the pathogenesis of aortic dissection is in fact rupture of the vasa vasorum in an aorta which has been weakened by medial degeneration. Medial degeneration is one feature of Marfan's syndrome, and patients with this syndrome are susceptible to aortic dissection. Medial degeneration is also seen in patients with hypertension, regardless of whether they have an aortic dissection. Obviously, many more people will have degeneration of the aortic media than will have dissections. Thus, a combination of conditions is required for dissection to develop: medial degeneration must occur and then there must be either a tear in the intima or rupture of a vasa vasorum to direct arterial blood under pressure into the defective media. Interestingly enough, so far as we know, atherosclerosis has no role in this process.

How do we explain this patient's pulmonary infiltrate and hemoptysis? There was congestion and hemorrhage in the lung parenchyma and also extravasation of blood from the aorta, as evidenced by the hemothoraces, and extension to the loose adventitial tissue of the pulmonary arteries and the pleural reflections in this area. There was a similar extravasation of blood into the parenchyma of the lung and thence to the bronchi, from which it could have presented as hemoptysis. Hemoptysis is rarely associated with aortic dissection. Analysis of 505 cases of aortic dissection revealed that the incidence of hemoptysis was only 3%-5%.<sup>2</sup> Nowhere in the article do the authors describe or propose a mechanism for hemoptysis, but it seems reasonable to assume that this kind of dissection into the lung

parenchyma could quite possibly account for it.

There were several incidental findings. There was extreme atrophy of the pancreas but, despite this, no apparent clinical evidence of diabetes. Another finding consisted of a small papillary adenocarcinoma of the thyroid, something that is seen in about 2% of patients at autopsy at the University of Virginia.

In summary, then, this patient had, as Dr. McGuire predicted, a type A aortic dissection.

DR. SCHORLING: The operation mentioned in the protocol was a thoracotomy which was performed while CPR was being continued. The dissection was found at surgery and CPR was then stopped. At surgery, tense blood was found in the pericardium and was evacuated.

DR. JOHN A. OWEN, JR.: At what point in her course was heparin discontinued?

DR. SCHORLING: Heparin was continued up to the time of death. The thoracotomy was performed while she was anticoagulated.

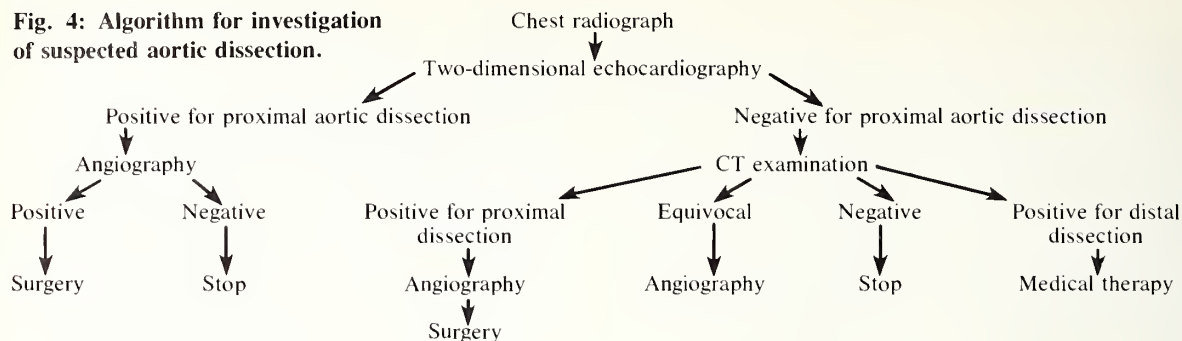
DR. ERIK HEWLETT: It is clear from the protocol that, while some of those taking care of the patient had considered in their differential diagnosis the possibility of dissecting aneurysm, that course was not pursued. One wonders how better this woman might have been evaluated, at least in terms of ruling out a dissection.

DR. GEORGE A. BELLER: There is an old dictum: as soon as you even entertain the possibility of a dissection in an acutely ill patient, you must rule it out. Often, dissection masquerades as other entities, as it did in this case. I am sure that Dr. Sturgill will confirm my impression that at least one dissecting aneurysm goes undiagnosed to demise in this institution every year and that this is true almost everywhere. As soon as one thinks of dissecting aneurysm, one should consider doing digital subtraction angiography, two-dimensional echocardiography or computed tomography to rule it out.

DR. HEWLETT: Dr. Dee has a particular interest and expertise in the diagnosis of aortic dissection, and he has some information on how to approach cases in which dissection is a possibility.

DR. DEE: I devised this algorithm (Fig. 4) now in use at this hospital. It is based on the assumption that surgeons will require an arteriogram before operating on a patient with a suspected dissecting aortic aneurysm. No one will dispute the fact that the patient should first have a chest radiograph. I am very impressed with the use of two-dimensional echocardiography in cases of suspected dissection.

**Fig. 4: Algorithm for investigation of suspected aortic dissection.**



It is a simple procedure, accomplished quickly. It is also portable and so could easily have been used on this patient in the intensive care unit—it stood a good chance of making the diagnosis in this case.

In the algorithm, I propose that 2-D echocardiography be used to triage patients either to angiography or to CT examination. If the echocardiogram suggests a proximal aortic dissection—that is, one likely to go to surgery—then the surgeon will probably want arteriography done; if the arteriogram is positive for a proximal dissection, surgery will likely follow. On the other hand, if the echocardiogram is negative for a dissection in the aortic root but aortic dissection is still suspected, the patient can then be referred for a CT examination.

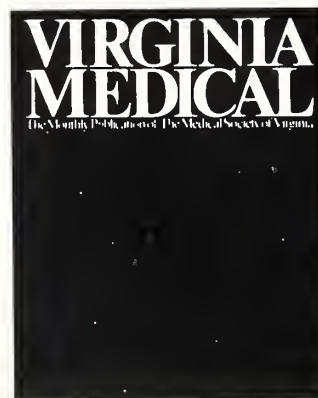
With CT scanning, there are four possible outcomes: 1) CT might be positive for a proximal dissection, in which case the patient will be rerouted to angiography; 2) CT might be equivocal, in which case angiography will be done; 3) CT might be negative, in which case no more diagnostic studies need be done; or 4) CT may be positive for a distal dissection, for which the standard treatment is now conservative medical management and medical therapy will be instituted (if a distal dissection requires surgery for any reason, arteriography can be done, of course).

I have been involved in a study at the University of Leiden in Holland where, for the past three years, routine CT examinations have been done on all patients admitted with the diagnosis, or the suspicion, of aortic dissection.<sup>3</sup> In every instance, diagnosis made on the basis of CT has been confirmed either by surgery or arteriography or both. Of 26 patients studied, five were deemed normal by both CT and angiography; we have followed each of them for at least one year and no untoward events have occurred, so it seems that these people were in fact normal. Two patients with dissection went to surgery on the basis of CT alone; in both instances,

the diagnosis made by CT was confirmed at surgery. In diagnosing proximal and distal dissections and in distinguishing the one from the other, both CT and angiography diagnosed all dissections. However, while CT diagnosed 12 proximal dissections, angiography diagnosed only ten and typed the other two as distal. It does appear that, in these two instances, CT was marginally better. And CT has the advantages of being a noninvasive procedure and one that can be carried out on fairly short notice.

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# Herbal Medicine

Robert Edgar Mitchell, Jr., MD,  
*Richmond, Virginia*

**T**HE BEST research laboratory may very well be a serendipitous one where the powers of observation are paramount, thus enabling the intelligent researcher to pick up on important surprises and make his own good fortune. It is this sixth sense which distinguishes the better investigators from the rest of us. Many such have gone before. It bodes well to think back on them.

From the brief notes to follow, as well as more intensive works extant on plants in medicine, we see that plants, from the earliest times, have provided man with real or imagined means of healing. This would date back at least 4,000 years, as tablets of stone have been uncovered which document findings to that time.<sup>1</sup> It would perhaps not be by chance, then, that two of the gifts of the biblical Three Kings are of plant origin, namely, frankincense and myrrh.

The father of the materia medica, Pedacius Dioscorides, a Greek army surgeon under Nero and author of *De Materia Medica*, described 600 plants in his treatises. Ninety of them are still in use today.<sup>2</sup> His botanical writings thus form the historic source of present day herbal therapy. For 1500 years, his was the most copied book in the field.<sup>3</sup>

A strong influence on Anglo-Saxon as well as other medical systems was exerted by Pliny the Elder.<sup>2</sup> Some of his aphorisms, however, were correctly challenged later by Leoniceus.<sup>4</sup>

The only Greek herbal of importance after Dioscorides was written by Galen. This great man, proponent of polypharmacy, is today best remembered in pharmacy for his "galenicals" (a decoction of vegetable drugs).<sup>4</sup> Galen was a most voluminous and ardent writer, composing 30 books on pharmacy alone! His fame persevered for several centuries in many medical disciplines in which he was the acknowledged leader.

Arabian pharmacy survived the eclipse of Arabian medicine in the 15th Century. This was largely done through contacts with far away peoples and

places. Among the drugs introduced at that time by Arabian pharmacists include senna, camphor, sandalwood, rhubarb, musk, myrrh, cassia, cloves, aconite, mercury, alcohols, aldehydes, syrups, and flavoring extracts such as orange, lemon, rosewater, tragacanth. Unfortunately, hashish came along at that time.<sup>1,2,4</sup> Thus we see that Arabian pharmacy and chemistry effects carried over into European materia medicas and eventually into our own, long after their political and military powers had waned.

Charlemagne is reported to have had a herb garden or "physic-garden".<sup>4</sup> These were monastic gardens, tended for the most part by monks.

Botanists of the Renaissance were remarkable for their ability to write and keep alive the investigative spirit needed to advance that important field. Otho Brünfels of Germany<sup>2</sup> exemplified this group. These men were largely influenced by Islamic methods as they set up physic-gardens and published prescriptions. An interesting interlude at that time was the so-called "doctrine of signatures". Botanists of the 17th Century proposed the tenet wherein plants were attributed to have curative properties based on their resemblance to the certain part of the human body that they were supposed to affect. One of the more interesting was described by William Cole, an Englishman, who felt the walnut to be a "signature of the human head and hence very helpful in assisting the brain to resist poisons". Heartsease, adder's-tongue, and maidenhair are legacies from that time. Fortunately, most physicians of the day did not subscribe to the "doctrine of signatures".<sup>2,4</sup>

The 17th Century showed great advances in modern medicine and botany. Botanical advances were made, for the most part, by men trained in medicine. Many of these men were accomplished artists and painted life-like portraits of the plant kingdom which helped spread this knowledge widely.<sup>1,2,4</sup> Great botanic gardens were started then (especially Oxford, Chelsea Physic, Edinburgh, and, later, Kew). Their agents travelled around the world to bring home interesting and exotic plants. That, of course, led to the work of the chemists and herbalists extracting active principles from the plants. Modern pharmacology was thus founded as these investigations went forth.<sup>1,2,4</sup> An incomplete list of important discoveries of active plant materials at this time includes morphine, strychnine, quinine, cocaine, aniline, atropine, conine, papaverine.

Also of note is that the 17th Century produced one of the greatest of physicians, botanists, chemists and physicists in the person of Hermann Boerhaave ("the Batavian Hippocrates"). His descrip-

Address correspondence to the author at 7601 Forest Avenue, Richmond VA 23229



*Baptisia tinctoria*



*Taraxacum dens leonis*



*Cornus florida*

tions of plants and drugs obtained from them were of enormous import. This famous man, perhaps best known for his original description of spontaneous rupture of the esophagus (Boerhaave's syndrome) in a Dutch admiral, is equally famous for his descriptions of medicinal plants and the rudiments of an understanding of their active chemical principles.

Of current interest is the knowledge that the Botanic Garden at Leiden<sup>5</sup> has replanted and restored the garden exactly as it was when Boerhaave himself was the professor of botany and director of the garden, among his other academic pursuits.

The settlements at Jamestown and other North American areas had these physicians continuing the European pharmacopias, namely, the herbals, using such as saffron, parsley, snake root, elder, rhubarb, opium and others in varying proportions and doses.<sup>1,3,4,6,7</sup>

Civil War restrictions in the South were overwhelming at times in many areas, especially for available effective medicinals. The Union naval blockade was disastrous in that respect. As a declining number of blockade runners made it through, bringing with them their precious cargoes, medicinals sorely needed by the military as well as civilian population became nearly non-existent. Clearly, an alternative means of obtaining these drugs was needed. This led the Surgeon General of the Confederate States of America, Dr. Samuel Moore, to institute a search for plants with medicinal value indigenous to the area.<sup>8</sup> *The Confederate States Medical and Surgical Journal* of 1864 describes some of these drugs. Among them are *Baptisia tinctoria* (for rattlesnake bites), *Papaver somniferum* cultivation (white poppies for opium), *Cornus florida* (dogwood bark used for malaria and a general fever remedy), *Smilax aristolachia* (sarsaparilla) and *Smilax china* (china root, for treatment of venereal disease), *Gelsemium sempervirens* (yellow jasmine, a plant from Hahnemann's original pharmacopia), *Eupatorium perfoliatum* (boneset, an old Indian remedy to break fevers, especially for malaria, typhoid, influenza), *Taraxacum officinale* (dandelion for kidney and bladder disorders), *Datura stramonium* (thorn apple, dried and smoked for bronchial asthma), and others.<sup>3,6,9</sup>

Our modern synthetic drugs have their origins in the hundreds of compounds whose structures were known by the late 19th and early 20th Centuries.

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The illustrations are reproduced from *Medical Botany*, by R. Eglesfeld Griffith, MD, published in Philadelphia by Lea and Blanchard in 1847. They appear here through the courtesy of the Virginia State Library.



This development of synthetic drugs was based on a desire to make analogues of medicinal compounds isolated from plants.<sup>3,4,6</sup> Drugs of this era include anti-pyrine, aspirin, barbiturates, chloral hydrate.

Domagk (Prontosil red, 1935), Fleming (penicillin, 1928), and later the combined chemical staffs of Merck (streptomycin), Lederle (Aureomycin®), and Parke-Davis (Chloromycetin®), with others following rapidly, led us into the antibiotic age. Compounds from molds and fungi are being tested continually, as well as semi-synthetic antibiotics currently being manufactured. It is fair to state now that this group of compounds (some of which were discovered by accident) will continue to be among our most important pharmaceuticals for years to come.

Much of the present search for useful plant compounds is concerned with anti-cancer agents and biologic response modifiers in patients with cancer. These herbal drugs may promote or enhance the natural host defense mechanism. The National Cancer Institute is continually screening thousands of plant collections for medicinally active properties. Among interesting substances discovered are vinblastine and vincristine.<sup>6-7</sup> The road to success with safety is long, tedious and difficult as well as expensive. It is important for us to realize that there is a proper place for plants in our armamentarium, since some of them are rich sources of compounds which cannot be manufactured inexpensively yet are essential in preparing certain drugs. A leading example of this would be yams, rich in diosgenin, from which hydrocortisone and related drugs, as well as most oral contraceptives, are manufactured.<sup>6</sup> Without yams (*Dioscorea* species) and similar type plants, the cost of these drugs would become enormous as animal sources of these hormones may be generally retrieved in small quantities only.

Another interesting observation is that the old herbalists seem to be completely vindicated in their dicta that a particular plant had to be gathered at a certain time of the year, as well as certain times of day or night, especially in view of our present knowledge of variations in a drug plant within a 24-hour period.<sup>3,6,7</sup> Selecting plants with care and removing them at a particular time and season would give a reasonably standardized dose, as shown by Withering's study of foxglove.<sup>6</sup> Circadian rhythm was known then even if less well understood than it is today.

For the heady medical traveller, being catapulted into the 21st Century with promises of ever yet more miraculous pharmaceutical triumphs, a little retrospection and even introspection may be in



*Papaver somniferum*



*Datura stramonium*



*Eupatorium perfoliatum*

order. It may be that we are even a little humbled to recall how some of our great drugs arrived on the scene. Certainly, all was not serendipity. Also we must remember that time is very short. We are having our lands blacktopped over and otherwise destroyed, our streams polluted, our wetlands destroyed, our air fouled. We must prevent this takeover of our vast natural dispensary if we are to preserve for future generations cures with the herbal drugs with which we currently abound and whose mysteries are being unlocked. Many plants used by primitive peoples as pain-killers, abortifacients, febrifuges and others also are being examined. Many more need to be. Our continued progress regrettably causes deleterious effects on our environment. Care must be taken as we expand, else we will lose forever many of our plant species and even plant families with potential medicinal value. A better life for future generations can be enhanced by keeping our precious plants safe and ensuring a foremost place for our herbal medicinal research. Surely, statistically, there must be many answers to present-day diseases and those yet to be discovered lying hidden in our fields and streams, waiting to be found for the benefit of all. The rape and desecration of our natural resources in the name of progress is wrong. As the Confederate Surgeon General determined long ago, an alternative route must be found. We must honor and preserve our indispensable Plant Kingdom. We cannot do less.

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## Infant Botulism in Virginia: Case Report

Ben D. Wilmot, MD  
*Alexandria, Virginia*, and  
Mhairi G. MacDonald, MD,  
*Washington, DC*

**T**HE first case of proven human infant botulism occurred in California in 1931.<sup>1</sup> However, it was misdiagnosed at the time. In 1974 nine patients were reported from Pennsylvania with a neurologic syndrome of then undetermined etiology which was almost certainly infant botulism.<sup>2</sup> The same syndrome was described in Southern California in 1976 but was not finally recognized as botulism until one year later.<sup>3</sup> The first report of correctly diagnosed overt infant botulism was that of Pickett et al, in 1976.<sup>4</sup> The purpose of this paper is to document the first case of infant botulism to be reported in the State of Virginia.

#### Case Report

A previously well, 5-week-old black female infant was admitted to a Northern Virginia hospital on February 23, 1983, with the history that she suddenly "forgot how to feed" approximately 24 hours previously and then developed progressive lethargy in association with "rattling" respirations. The infant was born at term, by planned cesarean section, with a birth weight of 3232 grams. Apgar score at 1 minute was 8, at 5 minutes 9. There were no postnatal complications. The infant was breast fed, with supplementary feedings of the soy protein formula, Isomil®, with iron, 2 to 3 times per week. There was a family history of milk allergy.

On admission, the infant was noted to be drool-

From the Department of Pediatrics, Georgetown University School of Medicine (Dr. Wilmot) and the Department of Child Health and Development, George Washington University School of Medicine and Health Sciences (Dr. MacDonald). Address correspondence to Dr. Wilmot at 1451 Belle Haven Road, Alexandria VA 22307.

This case was reported in summarized form in the January 1984 issue of the Epidemiology Bulletin, a publication of the Virginia Department of Health.

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ing, generally hypotonic, afebrile and lethargic. A working diagnosis of sepsis was made, and therapy with ampicillin and gentamicin initiated. While being taken to the x-ray department, she developed sudden respiratory arrest, requiring cardiopulmonary resuscitation. Subsequently she was referred to Children's Hospital in Washington, DC.

On admission to Children's Hospital, the baby was receiving ventilatory support. Response to painful stimuli included movement of the feet and an increase in heart rate. There was a weak gag reflex, but otherwise the baby was totally areflexic. The pupils were mid-size with a sluggish reaction to light. There was bilateral ptosis and generalized hypotonia, with occasional spontaneous movements against gravity. There were no focal neurological signs present, nor any other physical findings of note. Laboratory data revealed a blood glucose of 105 mg/dl, calcium 10.3 mg/dL, creatinine 0.5 mg/dl, blood urea nitrogen 12 mg/dl, and serum electrolytes within normal limits. Total WBC count 6,300/cu mm, hemoglobin 10.1 gm/dl, hematocrit 30.9%, 15% eosinophiles, platelet count 161,000/cu mm. X-rays obtained at the referring hospital revealed periosteal changes in the long bones of the extremities, which were interpreted as physiologic.

Antibiotic therapy was discontinued shortly after admission to Children's Hospital. Blood and spinal fluid cultures obtained at the referring hospital were negative. The infant's hospital course was characterized by variations in her neurologic status. Hypotonia was persistent; however, the gag reflex periodically disappeared, and movement against gravity was noted to be variable from day to day. Constipation was constant throughout the hospitalization, requiring saline enemas to obtain stools for culture and analysis for toxin. An electromyograph (EMG) recording one day after admission showed abundant small amplitude potentials of short duration, typical of botulism. A neostigmine challenge produced no improvement in her clinical condition.

Sixteen days post admission the infant still required ventilatory support but was showing daily improvement. All reflexes were present but weak. Three weeks after admission, ventilatory support was discontinued. One month from the day of admission, the baby was taking approximately 25% of her daily caloric requirement orally, and repeat EMG showed a ten-fold increase in the amplitude of muscle potentials to about 10–20% of normal.

The baby was discharged 5 weeks after admission, at which time she was still constipated and mildly hypotonic. She was feeding well.

#### SIGNS AND SYMPTOMS OF INFANT BOTULISM

Loss of facial expression  
Hypotonia  
Weakness in extremities  
Trouble swallowing  
Weak suck  
Constipation  
Depressed deep tendon reflexes  
Sluggishly reactive pupils  
Dilated pupils  
Altered cry  
Somnolence  
Extraocular muscle paralysis

Specimens forwarded to the Centers for Disease Control in Atlanta subsequently showed the serum to be negative for botulina toxin, as is expected in infant botulism, but the stool was positive for Type B botulin toxin and grew Type B *Clostridium botulinum* on culture.

After discharge from the hospital, the baby made good progress. She was constipated, however, for 6 weeks and showed some residual hypotonia at 3½ months. She progressed normally and at age one year was developmentally within normal limits, with no abnormal neurologic findings.

#### Discussion

This infant presented with a clinical picture typical of infantile botulism. The age of infants hospitalized with botulism has ranged from 3 weeks to 3 years, but it is most commonly seen in infants between one and 6 months of age. While classic adult botulism is caused by relatively large amounts of preformed toxin in improperly prepared foods, the infant syndrome occurs when spores of vegetative cells germinate in the gut and elaborate toxin. Resulting illness may range from inapparent to fulminant and can cause respiratory distress and death. Onset of the disease may be insidious, with constipation, mild lethargy, weakness and diminished gag reflex. There is also epidemiologic evidence that infant botulism may be responsible for some cases of sudden infant death syndrome.<sup>6</sup>

Various factors have been related to the severity of the disease and the rapidity of onset. Of the four types of toxin, A, B, E and F, which have been described in relation to human disease, A and B are by far the most common. Several authorities think that Type A produces a more severe and fulminant course than Type B, but there is considerable overlap in symptomatology<sup>5</sup>. In the United States, Type A infant botulism has most commonly been

reported west of the Mississippi, while Type B is more common east of the Mississippi.<sup>6</sup> There is some suggestion that breast feeding may offer partial protection against botulin toxin,<sup>6</sup> and the onset of disease appears to be more insidious in totally breast fed infants than in formula fed infants, particularly if the formula feedings contain iron. It has been hypothesized that human milk may contain antibodies to *C botulinum*, since adults ingest botulinum spores regularly. Iron is utilized by *C botulinum* for its metabolism.

Diagnosis of infantile botulism requires a high index of suspicion. Differential diagnosis includes sepsis, meningitis (infection and/or dehydration may be superimposed problems), organophosphate poisoning, myasthenia gravis, inborn errors of metabolism, and poliomyelitis. Preliminary diagnosis rests upon the characteristic history of onset and the clinical picture. Detection of *C botulinum* and/or toxin in the stool is confirmatory. Botulin toxin has never been detected in serum from a case of infant botulism. Electromyography may prove helpful in confirming the clinical suspicion of infantile botulism. However, not all patients will show the characteristic brief, small amplitude, over-abundant motor reaction potentials (BSAP).<sup>7,8</sup>

Once the provisional diagnosis has been made, the infant should be hospitalized for appropriate supportive care in an attempt to prevent respiratory arrest or aspiration. There is no proven value for the use of either botulin antitoxin or antibiotic therapy.<sup>9</sup> Use of aminoglycoside antibiotics is contraindicated because, as in our patient, this class of antibiotic when used in patients with botulism causes a potentiation of the neuromuscular weakness with subsequent respiratory failure or arrest. Apparently, aminoglycosides compete with acetylcholine for attachment at sites on the neuromotor end plate. This in combination with reduced acetylcholine release from presynaptic nerve terminals secondary to the effect of *C. botulinum* toxin, accentuates the muscle weakness to the point where effective function is no longer possible.<sup>10</sup>

Although initially well-publicized as a primary source of *C. botulinum* spores in affected infants, honey is probably implicated in less than one-third of infant botulism. The spores are ubiquitous and may derive from sources such as recently disturbed soil or vacuum cleaner dust. When spores are found in the environment, they have always matched the type found in the affected infant.<sup>6</sup>

Infant botulism is a potentially life-threatening disease and is probably more common than indicated by reported hospitalized cases. Cases at either

end of the spectrum of severity (very mild/lethal) may not be recognized due to a low index of suspicion. On the other hand, asymptomatic infant carriers have been described. In some cases, the severity of the symptoms will vary from day to day, possibly due to variations in the quantity of toxin released from the gut. However, the effects of botulinum toxin on the individual nerve endings are irreversible. The recovery process involves regrowth of terminal motor neurons, which seek out and innervate noncontractile muscle fibers.<sup>12</sup> Full recovery from the disease may take several months.

Since the signs and symptoms of infant botulism may suggest sepsis and/or meningitis, and because administration of aminoglycoside antibiotics in this condition may prove life-threatening, it is important to consider the possibility of botulism before initiating antibiotic therapy.

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# Surgical Management of Attic Retraction and Chronic Adhesive Otitis Media

Peter A. Wallenborn, Jr., MD,  
*Roanoke, Virginia*

**T**wo of the most frustrating conditions to treat in otology are attic retraction and the atelectatic middle ear, sometimes called chronic adhesive or constrictive otitis media.<sup>1,2</sup> In attic retraction, there is thinning of the pars flaccida and retraction of the drum over the malleus and the chorda-tensor fold. In the atelectatic ear, the drum is quite thin and transparent because of a loss of the membrana propria. It becomes tightly adherent to the promontory, and the incudostapedial joint appears skeletonized. Marked retraction often looks like a perforation.

Studies show that serous otitis media, with or without adequate treatment, may lead to severe ear problems. In one large intensive study of children with actively treated serous otitis media, Tos found that atrophy, retraction and tympanosclerosis were fairly common, accompanied by varying degrees of retraction of Schrapnell's membrane.<sup>3</sup> Atelectasis or adhesive otitis was found in 2.5% of the ears. Tos concluded that chronic middle ear disease stems directly from serous otitis media in childhood and that conditions arise that later stimulate the development of cholesteatoma, chronic adhesive otitis media and chronic otitis media with perforation of the tympanic membrane.

The use of tympanostomy tubes to provide prolonged middle ear ventilation has done much to prevent attic retraction and atelectasis of the tympanic membrane with its serious complications.<sup>4</sup> But in later stages, when the tympanic membrane becomes strongly adherent to the medial wall of the middle ear, this procedure is no longer useful. It

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Address correspondence to the author at PO Box 8306, Roanoke VA 24014.

does not restore the tympanic membrane to its normal position.

Otologists have been taught for years that there exists no satisfactory surgical treatment of an irreversible atelectatic middle ear. Despite many years of microsurgery, we were told that methods to correct this disease were disappointing and usually failed.<sup>2</sup> The general rule that surgery breeds more adhesions was thought to apply here. Otologists reported that despite careful dissection of the atrophic membrane from the medial wall of the middle ear and reinforcement with vein graft or fascia, there was usually an atelectasis and collapse of the middle ear to a degree worse than before surgery.

Sade describes four stages of tympanic membrane atelectasis: Stage I, mild tympanic retraction; Stage II, moderate tympanic retraction; Stage III, retraction of the tympanic membrane on the promontory; Stage IV, adherence of the tympanic membrane to the promontory and adjacent ossicles.<sup>5</sup> This paper describes techniques for surgical treatment of both early attic retraction and Stage III or Stage IV tympanic membrane atelectasis. The methods and techniques are not original with the author but are methods tried, proven and used for many years. The author has used these procedures eight years. Paparella in 1979 and 1981 outlined a method for correcting middle ear atelectasis similar to the one in this paper, but with minor variations.<sup>6,7</sup> Procedures similar to the author's for correcting attic defects have been reported.<sup>8,9</sup>

The techniques described in this paper follow the basic principles of reconstructive surgery of the middle ear outlined by McGee, namely: 1) To cover the tympanic space, there must be a functional tympanic membrane that is flexible, durable, and stationary. 2) The membrane must span a tympanum that is disease-free, lined with healthy mucosa, and containing air. 3) The eustachian tube must be functioning.<sup>10</sup> Cortical bone is utilized in reconstructing the attic defect. A fascia graft is used to reinforce and create a durable and stationary tympanic membrane. And Silastic®, which is inert in the middle ear, is used to provide an air-filled tympanum free of adhesions with healthy mucosa allowing access to the eustachian tube.<sup>11</sup>

## Surgical Technique

**Attic Retraction.** A postauricular incision is made, the periosteum elevated, and the outer table of the mastoid exposed. Using a diamond burr, a bone graft is outlined and then, using a mallet and chisel, a thin piece of bone is removed (Fig. 1). The

cortical bone is quite friable and must be handled gently. It is shaped under the operating microscope by holding it between the fingers and cutting the edges with the Steven's scissors or by grasping it with an alligator forcep and using the diamond burr (Fig. 2). Through the same incision a piece of temporalis fascia is harvested.

A transmeatal incision extending from 6 to 2 o'clock is used to expose the middle ear space and attic (Fig. 2A). The retracted area of the pars flaccida is completely elevated and the incus, malleus and chorda-tensor fold exposed. The sculptured bone is placed on the scutum and over the full area of the retraction pocket. A fascia graft is then placed over the cortical bone (Fig. 2B). The drum with the tympanomeatal flap is replaced in its normal position.

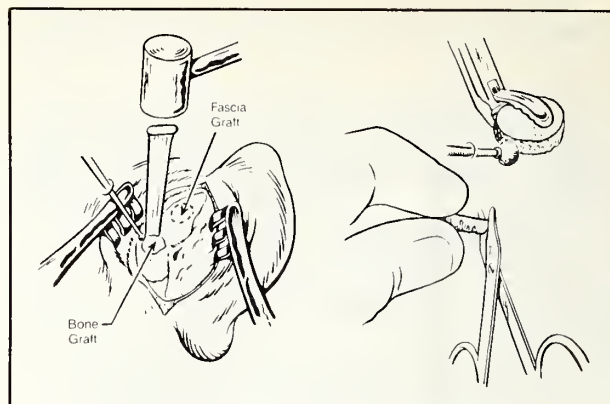
*Middle Ear Atelectasis.* Through a hockey stick incision above the ear, a large piece of temporalis fascia is harvested for grafting purposes. A transmeatal incision is made from 12 to 6 o'clock posteriorly and the annulus elevated (Fig. 3A).

The anesthesiologist is instructed to use 60% nitrous oxide and oxygen during the freeing of the atelectatic tympanic membrane, the difficult and tedious part of the procedure.

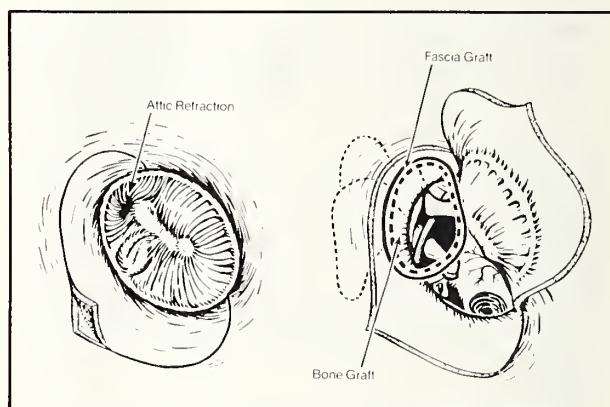
The dissection is begun between 6 and 8 o'clock. The drum is gradually lifted from the lower half of the promontory, using a small suction tip and a combination of blunt and sharp instruments; then the dissection is begun in the area of the chorda tympani nerve. The drum is slowly teased from the incus and stapes. From here the elevation is continued over the upper part of the promontory. Cutting the chorda-tensor fold is sometimes necessary to create an air space into the attic. This dissection inadvertently creates small perforations of the tympanic membrane. All adhesions and granulations are removed.

Mobility and continuity of the ossicles are evaluated. If necrosis of the lenticular process of the incus is present, this area is reconstructed. If ossicular fixation is present, this condition is corrected. Tympanosclerotic plaques are delicately removed. A tympanostomy tube is not used unless there is gross fluid in the middle ear. Of particular importance is that all squamous epithelium be removed from the middle ear space.

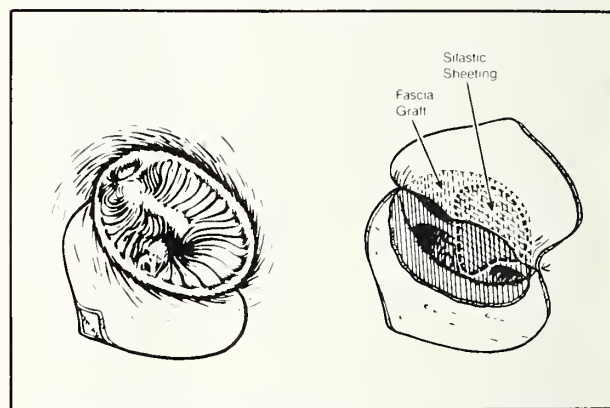
Silastic sheeting is sculptured to fit into the middle ear space over the promontory extending from the eustachian tube opening to the attic. The sheeting is placed beneath the annulus with no sharp edges or corners. A temporalis fascia graft is placed on the medial side of the tympanic mem-



Figs. 1A and 1B. Harvesting and shaping grafts.



Figs. 2A and 2B. Repairing attic retraction.



Figs. 3A and 3B. Repairing middle ear atelectasis.

brane and the middle ear is packed with Gelfoam®. The drum is replaced in its normal position, and the graft is positioned beneath the transmeatal flap (Fig. 3B). If the patient has attic retraction as well as middle ear atelectasis, both conditions are corrected simultaneously.

#### Material and Methods

Twenty patients comprised this study; ages



ranged from 4 to 56, the majority being under 20 years old. Four were females; 16 were males.

Twelve patients had retraction of atelectasis of the pars tensa, six patients had early attic retraction and two patients had both retraction of the pars tensa and the pars flaccida. Most patients had varying degrees of conductive hearing loss.

A number of patients had previous surgery related to their ear problems: 14 had a T & A; five a secondary adenoidectomy; nine a tympanostomy tube inserted; four a Type I tympanoplasty; and three a simple mastoidectomy.

Several patients had other surgical procedures performed at the same time: five patients had a simple mastoidectomy; one patient had a Reuter tube removed; one patient had a reconstruction of the ossicular chain; and one patient had a tympanostomy tube inserted.

The patients in this study had followups of 4-96 months, with the average being 37 months.

## Results

At surgery a great variety of pathology was found. Fibrous adhesions were encountered in all cases. Small tympanic perforations were found in eight patients. Six ears contained granulation tissue, and ossicular erosion, primarily involving the lenticular process of the incus, occurred in four patients. The drums of three patients were tympanosclerotic, and middle ear fluid was present in one patient. No cholesteatomas were found.

All patients had pre- and post-operative audiograms. Pure tone averages and pure tone air-bone gaps in decibels were used in reporting results. Patients are listed in two groups: Group A comprised 12 who had surgery for an atelectatic middle ear and two with surgery both for an atelectatic middle ear and attic retraction. Group B had six patients who had surgery for attic retractions only.

*Group A.* Preoperative audiograms showed hearing levels varying from 7-40 db with an average of 28 db. There was an average air-bone gap of 19 db.

Postoperative audiograms showed hearing levels decreased in three and improved in the ears of 11 patients. The average postoperative hearing level was 19 db, which is an average improvement of 9 db for each ear operated.

The average postoperative air-bone gap was 12 db, which is an average improvement of 7 db for each ear operated; 64% of the patients had a postoperative air-bone gap of less than 10 db and 79% less than 20 db.

Two patients have had subsequent procedures. One patient had a tympanostomy tube inserted, and

one patient had a revision tympanoplasty. The remaining patients have maintained good aeration of the middle ear space and have healthy appearing tympanic membranes. No Silastic implants or bone grafts have been extruded.

*Group B.* Preoperative audiograms showed hearing levels varying from 8 to 28 db, with an average of 17 db. The average air-bone gap was 13 db. There was no statistical difference between the pre- and post-operative air and bone hearing levels. This finding would be anticipated since early attic retractions have very little effect on hearing.

None of these patients had additional operative procedures. There has been no recurrence of the attic retractions and no absorption or extrusion of the bone grafts.

## Summary

Two of the serious complications of serous otitis media are attic retraction and middle ear atelectasis or chronic adhesive otitis media. Without treatment, these conditions usually lead to permanent hearing loss and chronic otitis media.

These simple surgical techniques correct a functional defect, improve hearing in atelectatic ears, and help prevent progression of middle ear disease.

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# VIRGINIA MEDICAL EDITORIAL

## Letter from a Doctor

Joseph Jones, MD  
Main Street  
Anytown, Virginia

Dear Joe,

I am pleased to report that I am doing well since my visit to your office just over a week ago. Your office staff was helpful and it was almost enjoyable for me to observe you doing your work with a light touch and a sure hand. Nevertheless, one aspect of my visit has continued to bother me a bit and I want to share my reservations with you. That one concern has to do with your decision not to charge me for my visit.

Not to charge fellow practitioners or their families has long been a fairly common courtesy. I suspect that this had to do with the relatively minor charges for services and practitioners' generally low incomes. As time went on, I suppose that referral patterns had something to do with continuing this practice, as better-paid consultants hesitated to charge their referring doctors and their families. My firm belief is that this practice has outlived its usefulness and is no longer good for the dispenser, the patient or their relationship.

The practice is not good for the practitioner for several reasons. It subtly says that the service is either worth nothing or that the usual price is out of line. It eventually leads to resentment, especially if one regularly cares for doctors who routinely refer paying patients to other practitioners. In addition, it sends the wrong message to the office staff regarding collection policies and establishes within the office the idea that there are two classes of patients.

The practice is not good for the relationship with the patient because patients do not feel free to "take your time" or tell about problems with their therapy.

Furthermore, it is not good in this time of increased cost awareness to insulate patients from the real cost of medical care. The reality is that there are costs involved in providing care that far exceed the doctor's salary: office rent, office staff, supplies and malpractice insurance, to name a few. To spread these costs over other paying customers to compensate for non-payment by colleagues is unfair to all.

Please do not take this as any sort of personal criticism. I think the practice of not charging fellow practitioners is firmly established in our society and I appreciate you giving me the opportunity to get on my soap box regarding this.

Yours truly,

Harry W. Easterly III, MD

## Letter From a Patient

Henry S. Campell, MD  
PO Drawer 3151  
Martinsville, Virginia 24115

Dear Campell,

For the last ten years you have attended to my physical medical needs but with decreasing attention to my psychological need to be somebody. It all started with your filing and computer billing system, which gave me an account number rather than just my name. Since department stores and credit



cards were doing it, I went along. Your nurse has always called me by name, but she got huffy the day that I forgot my appointment card with my file number on it.

You recall how sick I was in 1979 when you called in several consultants to help you with 'an interesting and perplexing case'? I was surprised to realize that I was neither a person nor a patient but a case. That I was an interesting and perplexing case, as you happily pointed out, did not help my self-esteem nor my faith in the humanity of my physicians. But you helped me to get well, and I appreciated it, and I overlooked my dehumanization.

When I had my relapse two years later, you sent me to the hospital for tests and x-rays. Once I had signed my name on some promissory notes and other forms, I lost my name and became "Next." I was amazed at how well the personnel could command my attention without ever calling me by name.

My drug store bill was huge, with all those modern medications which helped me get well. All of those prescriptions were numbered, and no number, no refill, regardless of whether or not I still knew my name.

You were so justifiably proud of your diagnostic

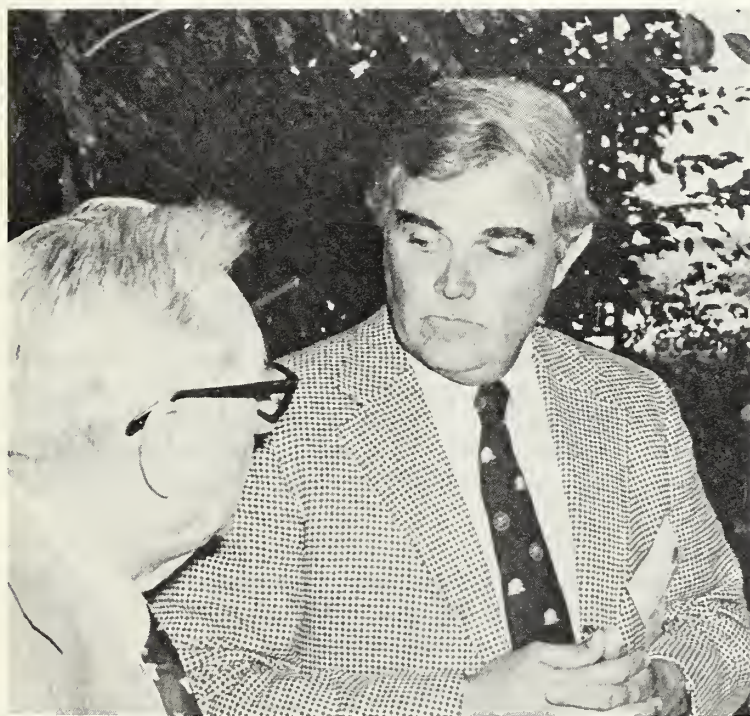
skill and treatment that you published my "case report." I still have a copy. Your opening sentence started thus: "This case report of a 48-year-old male individual. . ." I was no longer a name nor even a set of initials. Worse, I was not even a man, just a male individual. In that same journal were reports on young males, young females, IDMs, etc. Not one report called anyone a boy, girl, child, man or woman. (I finally discovered that an IDM was an infant of a diabetic mother.)

It may be petty on my part, but I have consulted with both my attorney and my psychiatrist about this loss of identity. My attorney tells me that I do not have a case until I get a mediocre or bad result. Then you will be a sitting duck. My psychiatrist suggested that I apply for a new set of numbers, but I think he was humoring me.

I do not wish to become even more dehumanized by biting the hands that have helped me to health, but I must be "me", a man, a Mr., or even Henry once people know me. I am not one to stand on ceremony, but I can no longer tolerate having just a number or being called a 48-year-old male individual. Just describe me as an old, tired, bald man, and I will be both grateful and happy.

Yours truly,

Henry Simon



## Dr. Caravati is new President Elect

Meeting last month in Williamsburg, Medical Society of Virginia members chose as President Elect Dr. Charles M. Caravati, Jr., Richmond, who is shown at left listening to Dr. Emmett C. Mathews. Elected as vice presidents were Dr. Joseph H. Early, Jr., Hillsville; Dr. William W. S. Butler, III, Roanoke; and Dr. Ira J. Green, Alexandria. Dr. Harry C. Kuykendall, Alexandria, was installed as President, as see page 718, and Dr. Richard L. Fields, Fairfax, and Dr. William H. Barney, Lynchburg, were reelected Speaker and Vice Speaker of the House. Va Med's January issue will carry pictures of the meeting.

# VIRGINIA MEDICAL OBITUARY

## Dr. G. S. Fitz-Hugh, MD

Dr. Glassell Slaughter Fitz-Hugh, professor emeritus at the University of Virginia in Charlottesville, died October 2 after a prolonged illness. He was 77.

A native of Charlottesville, Dr. Fitz-Hugh was graduated from the University of Virginia and continued through its School of Medicine, receiving his medical degree in 1933. After training in otolaryngology and ophthalmology, he became a member of the university's faculty and in 1951 was elected chairman of the Department of Otolaryngology and Maxillofacial Surgery. When he left the university for private practice in 1977, he was made professor emeritus, and the Fitz-Hugh Chair of Otolaryngology was established in his honor. He retired in 1983.

A member of many professional organizations, Dr. Fitz-Hugh had served as president of the American Laryngological, Rhinological, and Otological Society, the American Laryngological Association, the Virginia Society of Ophthalmology and Otolaryngology, and the Albemarle County Medical Society and had served as chairman of the Otolaryngological Section of the Southern Medical Association. For his alma mater, Dr. Fitz-Hugh had been a member of the board of managers of the UVA Alumni Association and was a past national president of the University of Virginia Student Aid Foundation.

## W. B. McIlwaine, MD

Dr. William Baird McIlwaine, who had practiced pediatrics in Petersburg for 55 years, died October 12 at Petersburg General Hospital. He was 92 years old.

Born March 9, 1892, in Petersburg, Dr. McIlwaine received a bachelor of arts degree from Hampden-Sydney College in 1912 and a medical degree from the University of Virginia School of Medicine in 1917. He interned at Kings County Hospital in New York City before joining the Army in 1918. After discharge from the service he worked at the Bellevue, Willard Parker, and Ellis Island Hospitals in New York, returning to Petersburg to start his pediatrics practice in 1920.

Dr. McIlwaine had been president of the Virginia

Pediatric Society and the Petersburg Medical Facility and had served two terms on the Virginia Board of Medical Examiners. He had belonged to The Medical Society of Virginia since 1917 and held long-time memberships also in the Southside Virginia Medical Society and the American Pediatric Society.

## Joseph W. Houck, MD

Dr. Joseph William Houck, retired Lynchburg surgeon, died September 8 at Virginia Baptist Hospital in Lynchburg. He was 78. Born in Harrisonburg, Dr. Houck was a graduate of Cornell University and the University of Virginia School of Medicine, where he also performed his surgical training. He was a veteran of World War II with the rank of Navy commander.

Dr. Houck had been a member of The Medical Society of Virginia for almost 40 years and belonged also to the Lynchburg Academy of Medicine and the American College of Surgeons.

## Memoir of W. F. Cavedo 1910-1983

*By William R. Hill, MD,  
Alvah L. Herring, Jr., MD,  
and William A. Young, MD*

William Fitzgerald Cavedo was born on March 14, 1910, the son of Lelia Britt and William Seabrook Cavedo, and died on December 20, 1983, at the age of 73. His father owned and operated Cavedo's Drug Store at the corner of Floyd Avenue and Robinson Street near Retreat Hospital in Richmond. This store was well known in the community and served the West End of Richmond for many years.

Fitzgerald Cavedo, better known as "Fitz", attended public schools in Richmond and was graduated from Richmond College, now the University of Richmond. He received his medical degree from the Medical College of Virginia in June, 1937. The next year, 1937-1938, he served an internship at the Medical College of Virginia. The next year he



served a one year surgical residency at Grace Hospital. Following he spent one year at New York Polyclinic Medical School in Operative Obstetrics and Gynecology. He entered general practice in Richmond and was on the staff of Grace Hospital. At that time it was the custom for doctors to make house calls and Fitz did his share. He was also the examining physician for the Local Board #1 of Richmond, and was awarded a certificate for this service in June, 1942. He was not eligible for military service but served very diligently on the "home front" where physicians were in great demand.

Fitz was well respected among his colleagues and enjoyed a large family practice until the last few years of his life. About 1971 Fitz suffered a stroke which left him partially paralyzed. However, after his recovery and with the help of a dedicated staff and wife he continued to do office practice until about 1979 when he suffered a second stroke. He closed his office and entered a nursing home in March, 1983, where he remained until his death.

Fitz was a member of the Richmond Academy of Medicine, the Country Club of Virginia and Hanover Avenue Christian Church.

He is survived by his wife, Amy Bumpass Cavado; his daughters, Dr. Barbara C. Worthington of Winston-Salem, North Carolina, and Mrs. Amy C. Swartz of Glen Allen, Virginia; one grandson, Tyler Clarke Worthington of Winston-Salem, and one brother, Willis Cavado of Atlanta, Georgia.

Fitz Cavado was a physician who never sought publicity but was dedicated to his patients and their well being. He will be missed by his colleagues, many patients and friends.

## Memoir of Cora Corpening 1892–1984

*By John A. Mapp, MD*

I had the opportunity and most enriching experience of being associated with Dr. Cora Z. Corpening, who died on September 1 in Virginia Beach.

She practiced in Virginia Beach for 40 years, until her retirement in 1965. Her latter years of active practice were my beginning ones, and she allowed me to associate with her. I soon learned something

about the art of medicine, above and beyond book knowledge. Some of her quotes bear repeating:

*I never lost a patient by asking for a consultation—only gained their confidence in me.*

*Never feel you have to write a prescription to treat a patient, even when pressured. A true patient will appreciate advice even more.*

*Don't be afraid to say "I don't know" to a patient. Being thoroughly honest with the patient only strengthens the doctor/patient relationship.*

This kind of advice is ageless and has always helped me. Now I pass it on to others when I can.

Born in 1892 in Caldwell County, North Carolina, "Dr. C" was the second female graduate of Tulane Medical School (1917) and was valedictorian of her class. Getting to that point alone took a lot of dedication. Medical schools didn't want women, and she was turned down at many schools. The University of North Carolina finally took her (it was then a two-year school) "on trial," and only after she appeared personally to appeal her initial rejection. Because she did so well there, Tulane took her for the final two years.

She then served internship and residencies in Norfolk (St. Vincent's Hospital), Suffolk (Lakeview Hospital), and New York (Bellevue Hospital). Her internship at St. Vincent's had to be one of the hardest years anybody could spend. The flu epidemic was on and almost all able-bodied physicians were in the service. Imagine working 365 days with no vacation and only a few weekends off in an overcrowded hospital, understaffed with physicians, and during the flu epidemic of 1917–1918. Dr. C was the only intern to complete the year.

In all, she practiced medicine for 50 years with hardly a break but found time to remain active in church and community affairs and raise a daughter, Jane Kibler. Finally, after retirement, she traveled extensively "to see all those places I thought about for years."

During my association with her, her peer physicians often talked about her in awe. One once told me, "She could write the book about the 'art' of medicine." Another said, "Whenever she sends me a patient, I always ask her what she thinks first, because she's usually right."

Without a doubt, her role model as a physician during her time would serve as a model for any time. We'll miss her.

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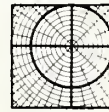


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